THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—23RD YEAR. SYDNEY, SATURDAY, AUGUST 29, 1936. No. 9.

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The E. C. Stirling Lectures.[1]

LECTURE I.

LOSS OF CONSCIOUSNESS DUE TO CIRCULATORY CAUSES.

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IT is probably impossible to say positively in many cases whether loss of consciousness is due to causes essentially concerning the neural mechanism or to changes in the composition and distribution of the blood. For example, there is a vascular basis for some forms of epileptic seizure, and indeed it may be an important factor in all epilepsies. In fact the more we consider the causes of unconsciousness, the more we realize the significance of the vascular factor: a study of ontogeny and phylogeny convinces us of this; the provision of nutriment is the first consideration, after that the regulation by some higher authority comes into the picture.

In considering this subject here I make no attempt to include all possible causes of loss of consciousness nor to describe them; I wish to draw attention to the more important from two points of view, that of diagnosis and that of the underlying mechanisms involved. Any attempt to understand these causes must depend in the first place upon physiological principles; of this we have a reminder in the title of these lectures, which commemorate the life and work of Edward C. Stirling, for many years Professor of Physiology in Adelaide, one of the great men of a prior generation, who has left his mark on medical science and education in Australia. I am conscious of the honour you have done me by inviting me to deliver these lectures, and I wish at this stage to pay tribute to the debt owed by the clinician to the physiologist.

[1] Delivered at Adelaide, May, 1936.

R. J. S. McDowall, Professor of Physiology in King's College in the University of London, has recently spoken in celebration of the centenary of the birth of Sir Michael Foster.[1] He remarks that "we see medicine and surgery claiming as their own methods of which many are the fruits of patient and ill-remunerated toil of the early pure physiologists". In these days, when pressure on the curriculum is forcing the pre-clinical studies into even smaller compass, let us not forget that we must remain students of physiology in direct relation to the practice of medicine. In the subject we now consider it is striking to observe that we halt in doubt or ignorance precisely where up to the present physiology has not yet fully instructed us.

Concerning the riddle of consciousness I shall say something later, but point out here that there is, as Hughlings Jackson[2] insisted, no such entity as consciousness; it is not a delimited function of the brain such as movement or hearing. We must recognize varying degrees of consciousness and its defects, but while discussing the circulatory type of faints it is well to have some standard. For this purpose we may adopt the definition of Ferris, Capps and Weiss[6] as being simple and compact: a "loss of contact with the outside world associated with amnesia and loss of voluntary coordinated muscular activity". It is natural that the loss of "the most special of all nervous processes" should cause alarm and even panic; in fact this feeling is doubtless biological in origin. Who has not observed or even felt that dread of a general anæsthetic, or known that last groping with the disorientated mind before the black tide of unconsciousness submerges everything that makes our own personality? This fear that

> some death
> Will run its sudden finger round this spark
> And sever us from the rest

is not a craven weakness, but an urge universal and inescapable. It is interesting, too, to witness how a person on the verge of fainting may, by a supreme effort of will, force back the wave which threatens to extinguish his control and volition, and may remain the director of his mental affairs, shaky but triumphant.

Loss of consciousness of itself is, of course, always a nervous phenomenon, but many of its most important causes are to be found outside the nervous system. These causes may be simple lack of nutriment to the brain, or some disturbance of the function of certain brain cells due to the presence or absence of some important chemical substance in the blood stream. Gowers[3] has pointed out that simple syncope from failure of blood supply cannot be immediately due to lack of blood cells *per se*, since the nutrition of the brain cells depends upon the composition of the lymph which bathes them; syncope is rather due to the suddenness with which the mechanical or physical relations of the blood supply are disrupted. It should be remembered that like any other organ the brain can accustom itself to extraordinarily unfavourable conditions if it be but given time to make adjustments; it is sudden disturbances in particular that endanger the continuity of brain function.

The most striking forms of loss of consciousness are seen in syncopal attacks, in which the result is sudden and dramatic; but in other conditions the onset may be much more deliberate.

Persistent or Overwhelming Toxæmias of Bacterial Origin.

Persistent or overwhelming toxæmias of bacterial origin are seen in cases of acute infection, for instance in typhoid fever, the cause being in part at least related to the circulatory function. The following may serve as a good example.

A girl aged twenty-two years was brought home from a brief holiday, having been ill for several days. On arrival she was found to be suffering from lobar pneumonia, but her general condition was so bad some hours afterwards that a consultation was thought advisable. When seen at this stage she was absolutely unconscious. There was obvious consolidation of most of the right lung, the temperature was high, over 40° C. (104° F.), the pulse rate was over 130, and the beat was very thin. The extremities were cold and spangled with cyanotic areas, and the patient seemed about to die. Looking at her, motionless and oblivious of everything, we felt that the prognosis was almost hopeless; but nevertheless she eventually made a good recovery.

Perhaps the transportation of this patient at an unfavourable period of her illness accentuated its severity, but her toxic coma was a striking one.

Other Chemical Disturbances.

Other chemical disturbances may occur, and may be positive or negative in nature. (i) Positive: (*a*) Drugs, of which anæsthetics form an obvious example. (*b*) Other substances abnormal either in nature or amount, may be present in the blood stream as the result of disease. Examples may be seen in uræmia, cholæmia and diabetic coma. In these cases the actual loss of consciousness is not extremely rapid as a rule, and may be preceded or accompanied by signs of irritation somewhat similar to those seen during the administration of some drugs such as ether. (ii) Negative. This type of change may be seen in anoxæmia or hypoglycæmia. An excellent example of the former is seen in carbon monoxide poisoning, in which the only harmful effect of the gas is due to its displacement of oxygen in the hæmoglobin.

A young man aged about twenty years was accidentally exposed to coal gas in a college bedroom for a number of hours. He was deeply unconscious, and although vigorous treatment including the use of carbon dioxide and oxygen soon replaced the carboxyhæmoglobin by the normal compound, he remained unconscious for nearly twenty-four hours.

This patient recovered completely, and it is interesting to record that there have been no sequels, though such signal anoxæmia which causes sufficient devitalization of the sensitive cortical cells with consequent prolonged coma, may also cause intimal spoiling in some of the arterioles, for instance in the basal gangliar region, with multiple minute vascular lesions.

Hypoglycæmia is well enough known also. Here the relation of cause and effect is simple and dramatic, and the final loss of consciousness may be startling in its rapidity, though the prodromal symptoms give reasonable warning.

Thus a diabetic patient who had brought his two children to have sugar tolerance estimations made was waiting on a seat

outside the biochemical laboratory, previously having, as was found later, hurried away from home *plus* his insulin but *minus* his breakfast. He suddenly fell forward in convulsions, but rapidly regained consciousness when glucose was injected into a vein. On coming to he was still grasping "in his hand of ice" not a "banner with a strange device" but a packet of sandwiches which, as a thoughtful parent, he had brought for his children.

Another young man was started on an insulin life as an out-patient. After the third injection (surely the dosage administered by his wife was sorely astray!) he lost consciousness rapidly, in this case also with convulsive movements. On admission to hospital his blood sugar was rapidly brought up to normal by the administration of glucose, but he did not regain consciousness till the next day.

All these cases strikingly show how the sensitive cerebral cells may swiftly fail in one of their most critical and delicate functions; and their recovery may be considerably postponed, for damage has been done and must be slowly repaired. Such recovery is not usually so slow as in the case related above, but prolonged coma following hypoglycæmia may occur as this shows. Other neurological sequels have also been described. It is most interesting to observe that the warnings given the patient of approaching hypoglycæmia are partly autonomic in nature (sweating, flushing, trembling *et cetera*) and partly affective (changes in demeanour and behaviour). The importance of the latter symptoms is that the patient, with his disordered mentality, may not perceive that anything is amiss.

Physical Disturbances of Circulation.

It is not necessary to dwell upon such important and familiar cases of unconsciousness as the vascular accidents, such as cerebral embolism, thrombosis or hæmorrhage. In intracranial hæmorrhage it is anæmia of the brain which chiefly produces the loss of consciousness, partly by disorganization of blood supply and partly by pressure. Mention may be made of the not uncommon subarachnoid hæmorrhage, often occurring in young people, and due to leakage from a congenital or acquired aneurysm on a cerebral vessel, usually on the circle of Willis. Unconsciousness may rapidly be produced in this way; in fact, as with intracerebral hæmorrhage, the patient may drop as though felled by a blow; the cause is readily revealed by finding blood in the cerebro-spinal fluid. Anything that will raise the intracranial pressure to a sufficient degree may disturb consciousness, particularly if the change is rapid. This is simply illustrated by the giddiness or even faintness which may be felt when forced expiratory efforts are made against resistance. Examples may be found in the giddiness caused by an undue strain to make any severe physical effort with the glottis closed, and in that giddiness which must be familiar to those who have tried to blow up the large balloons beloved of children of all ages on festive occasions. The curious "laryngeal vertigo" in which consciousness is lost after a sudden and unprovoked bout of coughing may also be mentioned; some of these cases are possibly of epileptic origin.

It is possible for unconsciousness to be produced by a slowly acting agent also; for instance, a cerebral tumour causing a block in the ventricular system. In this condition prodigious rises in pressure may occur before consciousness is disturbed, provided that the change takes place gradually.

One important clinical type of circulatory disturbance in the brain is that seen in the cerebral crises of hypertension, of which the following is an example.

A woman aged thirty-five years who had some family history of hypertension and who had had one miscarriage at four months was known to have hypertensive arterial disease. She had suffered from troublesome headache and giddiness for several days, when during one hot morning the headache became unbearable; she rapidly became unconscious, and showed slight general twitching. She was taken to a hospital, where no other definite evidence of nervous system involvement was found. The systolic and diastolic blood pressures were in the region of 190 and 110 millimetres of mercury respectively, and lumbar puncture yielded clear cerebro-spinal fluid under high pressure. After the puncture her condition began to improve, and within forty-eight hours she was dazed but otherwise well. She made a good recovery, and three years later was well enough to undergo a major operation without trouble, though she still remained hypertensive.

This type of occurrence is important, for there is no obvious disruptive lesion, and the prognosis is usually good. It will of course be understood that the disturbances mentioned here may be more or less gradual in onset, or they may be extremely rapid.

One very interesting form of unconsciousness is often seen in cases where Cheyne-Stokes breathing is well marked. This is rhythmic in nature. During the phase of hyperpnœa the patient may be conscious and talk breathlessly, but during the apnœic phase he mumbles into silence and lies in slumber until the breathing starts again. This waxing and waning of consciousness during periodic breathing is not uncommon, for instance, in cardio-vascular disease. It is extremely interesting, since it illustrates how rapidly the higher grades of cerebral function may be lost and regained provided too severe a nutritional change is not involved.

Syncope Due to Reflex Cause, with no Known Organic Factor.

Syncopal attacks may be and often are but slight in degree, or they may be profoundly alarming to all concerned. Faints are seldom directly due to disease of the heart, though it must not be forgotten that fainting may be an indication of some disturbance of or even severe damage to either the myocardium or what MacKenzie called the "genetic" system of the heart.

Obviously we must take all possible care that any existing heart disease is not overlooked; but it is a tragedy to label a reflex syncope as being due to an organic cardiac lesion. Medical practitioners frequently have young people brought to them who have fainted, and they have to settle the question as to whether the patient has a "weak heart". Almost invariably the correct answer is in the negative, yet even today examples are seen in which the patient has been given a solemn caution on the slender evidence of a dubious systolic murmur, an occasional extra-systole or even a forcible apex thrust which is felt over a wide area and is thought to betoken an enlarged heart. It is all the more vital, therefore, that we should understand clearly the common mechanisms by which fainting may take place.

The milder type of faint is exemplified in the following case, which is an instance of simple vaso-motor syncope of the postural type.

A girl, aged fifteen years, had an acutely inflamed appendix removed and made a good recovery. Several weeks later she complained that she had travelled by tram for the first time since her convalescence, and being unable to secure a seat was standing up. Suddenly everything went black and had she not been standing among other people she would have fallen. Since then she had felt faint if she stood up for long. On examination she appeared perfectly normal, but on trial after standing upright and quite still for several minutes she paled, staggered and would have fallen. Rest and graduated exercise soon restored her to a completely normal state.

For this condition of postural syncope ephedrine has been recommended.

In many cases of simple syncope there is no definite organic anomaly found in any system of the body. The familiar faint of emotional origin is of this type, but we should observe that several factors are often involved. The patient is often fatigued or otherwise not in good health, though this is not necessarily so. Physical indiscretions may pave the way for syncopal attacks.

A middle-aged business man, thick-set, fat and flabby of habit, had partaken too freely of the solid and liquid refreshments at a special luncheon on board ship. After lunch he was sitting under an awning smoking, was conscious of feeling the heat severely, became nauseated, and suddenly fainted. It was with great difficulty and only after considerable investigation, electrocardiographic and otherwise, that he could be convinced that he had no serious heart disease.

This case exemplifies the association of the fall in blood pressure with a dilatation of the splanchnic vascular network.

Athletes frequently faint after a strenuous contest; here the psychic factor of stress is also concerned—one of great importance. This is seen also when some unusual or disquieting experience upsets the person, such as the sight of an accident, or even being vaccinated. Again, such faints often occur in crowds where the air certainly is vitiated, but where, too, the emotional atmosphere is often one of tense excitement, as for example in religious revivals, and when escape is well nigh impossible. An interesting example of syncope is the following:

An intellectual and efficient young woman of twenty-two years complained of hay fever and naso-pharyngeal irritation. She was doing analytical work on castor oil seeds, and at my direction supplied me with material for making skin tests. Scratch tests were carried out with the crushed seeds, the husks and the fat-free benzene extract. About two minutes after applying the test materials to her arm I heard a sound, and looking up saw her diving headlong from her comfortable posture in a chair, to fall crumpled and helpless on the floor. She was pallid, with widely dilated pupils, the pulse was almost imperceptible and very slow, and her face and limbs were agitated by jactitating movements. When I found that wheals were already forming on her arm I felt a momentary fear that the attack was anaphylactic; but she recovered rapidly from what was a severe syncopal attack, though she was very shaky for some time afterwards.

Even the most experienced of us will admit that patients in the throes of a severe faint suggest the act of dissolution to an uncomfortable degree. The experience is also very disturbing to the patient, who may not completely recover for an hour or more afterwards. Even temporary blindness has followed profound or prolonged syncope.

The mechanism of these attacks is interesting. It is well known that great emotional exaltation may contribute to a suspension of consciousness. An over-stimulation of receptive centres has been suggested as the cause, but it seems more likely that there is an inhibition of higher cortical control.

The parallel in narcolepsy of the cataplectic attack may be cited here; to this I shall refer later. Surely there must be some direct action upon certain of the centres concerned with vasomotor control. Intense pain, as Gowers pointed out, may cause unconsciousness, apparently by direct vagal stimulation. But this is not all. Syncopal attacks are always rapid, but not of necessity sudden. Warning is often given; this takes the form of sweating, salivation and nausea, a feeling of sinking in the epigastrium (curiously reminiscent of the epigastric aura of epilepsy), even vomiting, and subjective or objective disturbance of the bowel. These happenings suggest vagal over-activity; so also do the slowing of the heart and the slow deep sighing respiration. Most important of all in actually causing consciousness to fail is probably the fall in blood pressure, which is usually conspicuous; when this occurs it is no doubt chiefly responsible for the deathly pallor of the face.

Lewis[4] and others following him call these faints "vaso-vagal attacks" and Lewis believes that they are caused by the reflex disturbances of a central nervous mechanism which is seen in action normally when the carotid sinus or depressor nerves are stimulated. The use of this name is most unfortunate; not because it is inappropriate, but because Gowers used it to describe a condition which is certainly quite different.

The physiological mechanism of syncope is of importance; recent work suggests that it is not necessarily the same in all cases. Weiss and Baker[5] have shown that mechanical stimulation of the region of the carotid bulb does not produce an invariable result. There may be several different types of cardio-vascular response to this reflex stimulus: the pulse rate may be slowed with or without fall of blood pressure, or there may even be a period of asystole; there may be distinct drop in blood pressure without slowing of the heart; there may be an intense pallor of the face, followed by a period of flushing, without substantial changes in heart rate or blood pressure; or there may exist combinations of these. The existence of arteriosclerosis or hypertension enhances the fall in blood pressure, and coronary disease predisposes to slowing of the heart. All these forms of vascular response may be associated with syncope.

Ferris, Capps and Weiss[6] in further work have studied a series of patients showing hypersensitivity of this carotid sinus reflex. Such patients suffered from spontaneous faints of fairly brief duration in which there was occasionally an aura, which were influenced by posture, and which could be reproduced by pressure over the carotid bulb but not over the artery below this point. In these artificially produced faints it could be shown that syncope did not always depend directly upon blood pressure changes or slowing of the heart; apparently they were sometimes due to a direct cerebral reflex, initiated by an unusually labile carotid sinus nervous apparatus. In a few such patients pathological changes, such as

dilatation of the bulb, or even a small tumour, have been demonstrated at the carotid bifurcation. There is some reason for believing that when the direct cerebral reflex causes vasoconstriction in the brain it is paralleled in some measure by the constriction actually seen in the vessels of the pallid face. I shall refer later to the possible significance of pallor in *petit mal*, and point out also that there is a correspondence between the behaviour of the facial and cerebral vessels following injection of histamine. (Weiss, Robb and Ellis.[7])

In this experimental study additional proof has been afforded of the fact referred to above, that it is the speed with which cerebral ischæmia is produced that is the most important factor; the cerebral symptoms are precipitated not by the degree of blood starvation, but by the rapidity with which the change takes place.

It certainly seems remarkable that such a reaction as reflex syncope should occur in a body presumably otherwise healthy. Is there some factor as yet unexplained which makes some persons more liable to faints than others? It is interesting to take the example of aortic regurgitation; in this condition faints are not uncommon, and they are of just this type. Here there are certain predisposing factors, namely the labile pulse pressure, the capillary dilatation and general disorganization of the intimate circulation of the tissues consequent upon the aortic leak; these are probably just as important as the leak itself. It is possible that hyperexcitability of the carotid sinus reflex may be of some importance in these cases. Clinical experience would certainly suggest that there is some other intrinsic factor in syncope.

Collier,[8] while holding strongly that syncopal attacks are in no sense related to epilepsy, believed that they should be considered to be of the same order as regards the pathogenic mechanism in the nervous system. Why, as he asked, should some persons be subject to this fall in blood pressure with all its related phenomena? He further suggested that a second or epileptoid factor might be necessary to produce convulsions in syncope. I have been rather fascinated by this suggestion, for I have seen some people lie stricken and immobile, a picture of vasomotor shock; others even with an attack of lesser duration may twitch perceptibly. I am aware that these speculations answer none of the questions raised, but probably certain affective reactions may more readily arise in certain persons; having arisen, they may vary in their capacity to inhibit normal control and balance of the vasomotor system; and the syncopal attack being once unleashed, the outward and visible results may also vary in different individuals. The gift to coordinate the contraction and relaxation of opposing muscle groups varies in different people, for example in good and bad tennis players or pianists; perhaps the compensating mechanism between the higher cortex and the vegetative centres and relays in the interbrain varies also.

Before proceeding it is of interest to consider briefly the mechanism of vasomotor failure in shock due to various pathogenic causes. Following in the main the classification of Blalock,[9] we recognize shock as being hæmatogenic, neurogenic, vasogenic and cardiogenic. In the hæmatogenic type the principal change is diminution of blood volume; the vaso-constrictor adjustments try to keep pace with it, so as to keep the blood pressure at an efficient level, but at last they fail, and the tissues starve and are poisoned. In the neurogenic form a primary vaso-dilatation occurs, which is due to trauma or other reflex causes; in the primary vasogenic type the action is chiefly on the blood vessels, as in histamine poisoning, and the rare cardiogenic type is best illustrated by the embarrassment caused by some influence such as the rapid filling of the pericardial sac with blood after an accident. With this classification in mind we may turn to the next group of causes.

Syncope Due to Reflex and Nutritional Causes when a Known Organic Factor Exists.

Syncopal attacks due to reflex and nutritional causes, in the presence of a known organic factor, are seen after severe injury, in surgical shock, in extreme exhaustion, in lack of blood (as in severe anæmia due to blood disease, or to rapid blood loss), in pregnancy, in acute infections, in certain cases of poisoning and in Addison's disease. It will be seen that some of these are indistinguishable from the first group; certainly as regards mechanism they are practically identical. Clinically there are some interesting examples here.

A woman, aged forty-three years, was suffering from Addison's disease; she had the usual signs, such as extremely low blood pressure, pigmentation *et cetera*, and complained of nausea and oppressive weakness. It was almost impossible to move or examine her without causing the subjective sensation of fainting or even a slight though definite syncopal attack.

The remarkable ease with which these attacks may be induced, especially during an exacerbation, is a notable feature of this disease. Those who have seen Somerset Maugham's play "The Sacred Flame" will remember an example of syncope in pregnancy in which the psychic factor of strong emotion in painful and difficult circumstances was an additional influence.

Sudden blood loss is a most important cause, especially when the bleeding occurs into the alimentary canal, and in many cases, when the intraabdominal pressure has been suddenly lowered by evacuation of the bowel contents.

A woman, aged forty-nine years, suffering from hypertensive arterial disease following an earlier toxæmia of pregnancy, felt sick during the night and had a desire to evacuate the bowels. She rose from bed and suddenly fainted, and was found white and almost pulseless. She soon recovered, and vomited a quantity of dark brown material, and next day passed a quantity of altered blood. She made a good recovery from what was undoubtedly a large bleeding from a gastric erosion.

A dentist in middle life had suffered from some dyspepsia, and had not felt well for twenty-four hours. He kept his usual appointments, and was actually at work when he felt ill and suddenly collapsed. He was seen by his medical attendant, who found him pale and shocked, with a low blood pressure and a heart rate which persisted at a rate of about 40 per minute. This was such a conspicuous feature of his state that his doctor actually suspected him of having heart block; it was, however, certainly due to a vagal bradycardia. When seen next day his obvious anæmia pointed to the diagnosis, and his passing of a copious melæna proved him to be suffering from an acute blood loss; it was due to a duodenal ulcer.

The occurrence of a slow pulse following a rapid hæmorrhage should be noted. In these cases it is not rare, though conventional teaching stresses the rapidity of the pulse in such circumstances. Syncope in bleeding duodenal ulcer, as all medical writers remark, is not uncommon; it should always be borne in mind.

I now digress from the peripheral circulation to consider the third category.

Syncope Due to Cardiac Disturbance or Cardiac Disease.

Though really falling into the previous category, syncope due to cardiac disturbance or cardiac disease merits separate consideration. Fainting in aortic regurgitation has already been mentioned; it is not truly cardiac at all in many cases. Much more important is fainting due to coronary occlusion. In this dangerous accident a faint may be the first symptom, or it may occur, and even recur, after a conventional initial occlusive attack.

A woman of fifty-two years was standing at a fence talking to a neighbour. Suddenly she felt ill, turned pale, and collapsed; she was found to be unconscious, looked extremely ill, and was carried into her home. She regained consciousness in part at least, and remained in a state of collapse or some time. She was breathless and shocked and was in bed for several weeks, during which time she suffered discomfort in breathing, especially when lying down, but had no pain. On recovery she was extremely weak and readily fatigued, and was found to have a slight anæmia of secondary type. Rest and administration of iron restored her to fair health. She could undertake no exertion without breathlessness, and began to have attacks in which she was faint, pallid and sweating, was intensely anxious, and felt as if she were dying, but still had no pain. She showed evident signs of circulatory distress; gradually congestive failure supervened, and after a partial recovery she sat up in bed one morning, gasped, and died instantly.

Here was a case of coronary occlusion without pain, but with severe syncope as its first symptom.

In other cases of classic occlusion it is found that for several days the patient has repeated attacks of swooning. These are certainly not necessarily due to successive infarctions, as is often shown by the patient's subsequent progress; such attacks are surely vagal in nature. It will be recalled here that exaggerations of sinus arrhythmia and sinus bradycardia are both common following coronary occlusion; undoubtedly these are also manifestations of vagal over-stimulation.

Much more obscure are those forms of syncope associated with disturbances of cardiac rhythm. Fainting may occur when the heart begins to beat too fast, or when it begins to beat too slowly, or when it temporarily ceases to beat.

Paroxysmal Tachycardia.

Paroxysmal tachycardia, a most interesting cause of faints, is not always recognized. Sutherland[10] pointed out that it occurs in children, and should be remembered as a cause of fainting in them. Why syncope should occur is not obvious. A paroxysm of tachycardia may last for days, and a feature of such an attack is the remarkable tolerance shown by both the patient and his heart; the patient recovers from the profound exhaustion and the heart from the dilatation with surprising ease and celerity. But it is at the moment of onset of the new rhythm that fainting may occur; all-important is this element of sudden change in producing syncope.

An interesting case is the following:

A young man, aged twenty-seven years, was sent to hospital with a provisional diagnosis of minor epilepsy. He had several attacks of sudden giddiness and faintness, had lost control of his faculties, and had a couple of times collapsed for a brief period. Some time elapsed before he could proceed or work after these happenings, but careful inquiry revealed that he definitely felt faint and giddy first. Sometimes he could sit down and rest; he was conscious of a disordered sensation in the chest, but had difficulty in helping himself.

These attacks proved to be bouts of paroxysmal tachycardia. The diagnostic fallacy is worth some emphasis.

A woman, aged thirty-five years, had similar attacks. During examination she was raised to a sitting posture on the couch; she immediately felt faint, and her heart was observed to double its rate almost exactly. An electrocardiogram showed this to be a simple tachycardia, and yet the inception of this more rapid rate caused some subjective faintness. At the beginning of her true attacks of paroxysmal tachycardia she suffered a more definite degree of syncope.

Such symptoms may occur not only in tachycardia of auricular origin, but also in the less common true ventricular tachycardia, particularly if the ventricular rate becomes extremely rapid.

Other Disturbances of Cardiac Rate or Rhythm.

The onset of bradycardia may cause syncope. So, too, may the inception of paroxysms of irregular rhythm, particularly auricular flutter. It has been suggested that the syncope in this case may be due to a momentary attempt of the ventricles to follow the rapid auricular rate, but electrocardiographic studies do not appear to support this idea. It seems more likely, as Sutherland says, that there may be a temporary lack of blood in the heart at the moment of onset.

Ventricular fibrillation, usually the precursor of death, will also cause cerebral anæmia and syncope by reason of the inefficiency of contraction of the ventricles.

Asystole in Heart Block.

Asystole in heart block is well known as a cause of fainting, chiefly in association with definite myocardial disease, though it may also arise through disturbance of the depressor nerve mechanism.[12] It is interesting that here the sequence of loss of consciousness, apparent suspension of animation and epileptiform twitchings is followed according to the length of time during which the blood flow to the brain is arrested. If the ventricles cease to beat for a few seconds only, consciousness is lost; the same happens if the rate becomes unduly slow for a few seconds. The exact conditions necessary to produce syncope or, in particular, convulsive seizures seem to vary with the individual. Here, of course, the sufferer is not young, and the cerebral vessels are usually more or less degenerate. The following case is instructive.

An elderly woman was alleged to suffer from epileptic fits. She had fallen on several occasions, and once cut her scalp severely on a brick path while walking in her garden. An actual attack when witnessed proved to be due to ventricular asystole, as was fairly obvious as soon as the extremely slow heart rate was noticed. She continued to have typical Stokes-Adams attacks till her death, which followed a succession of these seizures.

It is interesting to record that this woman had been known to suffer from a mitral stenosis all her married life, and that when a girl she had been ordered to an inland climate on account of hæmoptysis. It is curious that in all probability the original rheumatic lesion in her heart gave rise (perhaps with some plausibility) to two such aberrant diagnoses as pulmonary tuberculosis and epilepsy.

Before leaving the subject of cardiac disorders let me once more lay emphasis on the concept that it is the suddenness of a change in the cerebral circulation that upsets the balance; this principle of course governs many pathological symptom complexes seen throughout the whole realm of clinical medicine.

Other Forms of Vascular Syncope.

In dealing with other forms of vascular syncope we have a less clearly defined field of conditions which do not quite fit into any of the previous categories, and in it I place seizures of the type described by Gowers as "vaso-vagal attacks", though it is doubtful whether they are primarily vascular.

Gowers,[11] who was a most accurate observer with a most orderly mind, described cases in which peculiar attacks occurred, which he labelled with the not altogether suitable name of "vaso-vagal attacks". Indeed he was constrained to enter the correspondence columns of *The Lancet* in defence of this title, but admitted that he did not assign a definite anatomical basis to the word "vagal"; by its use he meant simply to indicate that the subjective phenomena noted in his patients were referable to the field of the vagus. Other writers have suggested, however, that some disturbance of the vagal nuclei actually does occur. It is obvious that Lewis and other writers, particularly those who have a cardiological bias, use the term "vaso-vagal" to describe ordinary attacks of syncope; and though, as pointed out above, the term is as a name quite justified, it is a pity that it has been so employed, as a great amount of confusion has arisen thereby. It is equally evident that Lewis[12] does not necessarily include in his account of syncopal attacks what he describes accurately as "Gowers's syndrome" and "Nothnagel's syndrome". Nothnagel described attacks in which the patient blanches, the limbs become cold and numb, the pulse is small but unchanged in rate and there are uncomfortable or alarming symptoms of anxiety, palpitation and giddiness. His cases were, however, of a restricted and unusual type. Gowers, who remarked that the condition he described was apparently the same as the "medullary syndromes" of Bonnier, has given a description which is characteristically vivid and accurate. The patients are frequently women, the attacks are subjectively and sometimes objectively alarming, and though sudden of onset, are usually more or less prolonged, lasting up to half an hour. These seizures may recur over a considerable period of years in some instances. At the onset there is usually some epigastric discomfort, once more recalling the epileptic aura, and an initial feeling of oppression may be followed by a conscious disturbance of the stomach and intestines, though vomiting does not occur. Then ensues more or less acute discomfort in the precordial region; the patient feels as if the heart were bounding or even stopping, and there may be some definite thoracic pain extending up into the neck. The limbs are cold and tingling owing, as Gowers suggested, to vascular spasm, and the patient may shiver. A most remarkable symptom stressed by those who have paid special attention to this syndrome is an unusual mental state, in which the patient finds it difficult to concentrate and to attend to what is said; speech is slow and there is an external impression given as of fatigue.

Loss of consciousness has occurred, but this is rare, and it may be asked why we should trouble here about a condition not usually associated with true fainting. I lay some emphasis on this because it has been the subject of great confusion and because the alteration in mental condition represents a step along the road to disturbance or even loss of consciousness. Ryle,[13] who has given an excellent description of these seizures, calls attention to the *angor animi*, which is a striking symptom. He points out how remarkable it is that in the absence of any evidence of cardio-vascular disease we should have so alarming and prolonged a disturbance, for alarming it is with the pallor, coldness and slow pulse, some degree of pain in the precordial region, the prostration, the shallow rapid respirations, and occasionally slight degrees of minor tetany. But, as he points out, the disordered sensation is not truly a fear of death as with true angina, nor is it quite a feeling of faintness, but perhaps it may be a variant of the "fading away" sensation felt by some patients with labyrinthine vertigo. If this be accepted, it is possible to link up these cases with those of migraine and other similar disturbances in which there is definitely a suggestion of interference with the higher grades of consciousness. Gowers, as a neurologist, perceived the possibility of certain of these cases being actually akin to epilepsy, a shadowy relationship which has since been exaggerated by some writers of text-books.

A woman, aged forty-four years, had suffered for over a year from attacks in which she had felt pain in the left breast and left arm, they often came on at night and were unrelated to effort. The whole of the left side of the body felt cold and she had feelings of "fainting away" and apprehensiveness which lasted for some time. Thorough examination showed no abnormality in any system. She described a "leaping" of the heart during attacks, but no extrasystoles were discovered, and the electro-cardiogram was normal in type. In her case, as in most of this type, there was a definite element of nervous stress due to family trouble.

Note how this history suggests the so-called "pseudo-angina"; surely many cases described as "pseudo-angina" and coronary spasm are of this nature. This is one of the instances where we should not be satisfied with a name; if we use the name "vaso-vagal" at all to denote these attacks it is better to call them "the vaso-vagal attacks of Gowers", so as to make it clear that a simple vascular syncope is not intended.

Lastly I would point out how fascinating a study of these curious symptoms may be; it shows how close is the bond between heart and mind, using these words in the actual physiological sense. This bond serves as a connecting link between the two branches

of our subject, and we next turn naturally to the nervous system in an attempt to wrest from it not indeed the secret of consciousness, but at least some knowledge of the disturbances in it that may give rise to a loss of our most essential function—that of ordered and independent being.

References.

(1) R. J. S. McDowall: "Sir Michael Foster", *The Post Graduate Medical Journal*, March, 1936, page 78.
(2) "Selected Writings of John Hughlings Jackson", Volume I, 1931.
(3) W. R. Gowers: "Faints and Fainting", *The Lancet*, March 2, 1907, page 565.
(4) T. Lewis: "Diseases of the Heart", 1933, page 98.
(5) S. Weiss and J. P. Baker: "The Carotid Sinus Reflex in Health and Disease", *Medicine*, Volume XII, 1933, page 297.
(6) E. P. Ferris, R. B. Capps and S. Weiss: "Carotid Sinus Syncope and its Bearing on the Mechanism of the Unconscious State and Convulsions", *Medicine*, Volume XIV, Number 4, December, 1935, page 377.
(7) S. Weiss, G. P. Robb and L. B. Ellis: "The Systemic Effects of Histamine in Man", *Archives of Internal Medicine*, March, 1932, page 360.
(8) J. Collier: "The Lumleian Lectures on Epilepsy", *The Lancet*, March 24, 1928, page 587; March 31, 1928, page 624; April 9, 1928, page 687.
(9) A. Blalock: "Acute Circulatory Failure", *Surgery, Gynecology and Obstetrics*, Volume CLVIII, March, 1934, page 551.
(10) G. A. Sutherland: "Fits and Faints", *The Lancet*, June 4, 1927, page 1271.
(11) W. R. Gowers: "Vagal and Vaso-Vagal Attacks", *The Lancet*, June 8, 1907, page 1551.
(12) T. Lewis: "Lecture on Vaso-Vagal Syncope and Carotid Sinus Mechanism", *The British Medical Journal*, May 14, 1932, page 873.
(13) J. A. Ryle: "The Study of Symptoms", *The Lancet*, April 4, 1931, page 737.

RADIOLOGY AND HEART DISEASE: THE MEDICAL ASPECT.[1]

By KEMPSON MADDOX, M.D. (Sydney), M.R.C.P. (London).

FOR over thirty years French and German cardiologists have made daily use of the radiological visualization of the living heart. American and Austrian workers have since added to the sound framework of their predecessors. British physicians, ever jealous of their reputation for extraction of the maximum information by the use of the unaided senses, have really only during the last five years added routine X ray examination of the heart to their diagnostic equipment. This conservatism is exemplified by the fact that up to the present time no text-book on the subject has been prepared by a British author, though large volumes are available in both French and German.

Our discussion tonight, however, has been foreshadowed by an identical combined meeting at the annual meeting of the British Medical Association at Dublin, 1933, to discuss precisely the same subject. I would commend you to the most excellent introductory papers by Dr. Parkinson, Dr. Kerley and Dr. Bramwell, which were delivered on that occasion, and which may be said to have strongly fanned the enthusiasm for routine radiological investigation which is so noticeable in London heart clinics today. With us, as far as my own observation goes, it is the exception rather than the rule, for the physician either directly or indirectly to employ X rays for cardiological diagnosis, except when disease of the great vessels is suspected. It is, of course, hopelessly impossible to encompass the title of this paper in a single lecture. It would be as easy to teach the complete art of auscultation in a single evening. It requires at least a month's post-graduate tuition before one's observations become reliable. Such a course is worthy of consideration by the Post-Graduate Committee in Medicine. All I propose to do is to indicate an answer to the clinician's question: "What further information may I expect to gain by submitting my patients to a routine radiological observation?" The technical and descriptive aspects of the subject I leave in the capable hands of Dr. Voss, although some overlapping is unavoidable.

An experienced physician, having compiled a careful and adequate history, and following a full clinical examination, often forms an opinion so accurate and informative that nothing whatever is added from instrumental or radioscopic examination, at least, say, in 80% of the cases. The remaining 20%, however, prove to be instances in which something more is gained from such methods of precision, something which illuminates the ætiology, pathology, or prognosis, or even rudely disturbs a presupposed concept. Exactly as the clinician must understand the indication for, and evaluation of, laboratory tests, he must also know definitely when and how assistance can be gained from the use of the X ray tube. Further, he is in the best position himself to search for confirmation or otherwise of his clinical diagnosis, and to peer for other findings in sites to which a proper knowledge of pathology and of the natural history of intrathoracic disease lead him. Without trying to be controversial, and with a full realization of the aid which is constantly forthcoming from our radiological colleagues in this field as in every other, I want to say most definitely that the cardiologist should make his own observations. He should do so alone, and not in the immediate company of the radiologist, though both may, with advantage, observe consecutively. With increasing experience in a small field he becomes a continually more skilled observer, forming reliable, detailed and independent opinions, just as the chest physician, the urologist or the neuro-surgeon does in observations on similarly limited regions. Further he should try to be equally as versed in pulmonary radiology as the chest specialist. He can hope to reach an efficiency at all comparable to that of such colleagues only by almost daily practice with the fluoroscopic technique.

The X ray screen has established itself as indispensably as the electrocardiograph in the armamentarium of the cardiologist. No cardiological investigation can be considered complete without its inclusion. Indeed, one would go so far as to say that if one had to choose between the results of percussion, auscultation, cardiography and fluoroscopy as supplying the nearest approach and the shortest cut to an accurate final diagnosis, one would choose the last mentioned. It is a universally observable fact that the most experienced cardiologists of the world are the most enthusiastic fluoroscopists, and many will withhold a final opinion until after the screen examination has been made.

I have said something of the clinician as radiologist, but so far nothing of the radiologist as clinician. Radiological observation is undertaken by the Röntgenologist when X ray facilities are unavailable to the clinician. Too often clinical notes are sparse

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 30, 1936.

or missing. Details such as age, height, weight, occupation and previous illnesses must be also systematically noted, as they are essential to accurate interpretation in the present instance.

In private practice most radiologists will make an adequate screen examination before taking a picture, but in hospital practice they are too often asked for a helpful opinion when they have never seen the patient at all, either in front of, or behind, the yellow glass. To rest content with the report, made on a single antero-posterior film, of "slight general enlargement" is fair neither to the radiologist nor to the patient. Fluoroscopy is the technique of choice for the clinician's use.

Fluoroscopy causes distortion, it is true, and permits of no accurate mensuration, but with the patient centrally placed, close to the back of the screen, the error is relatively slight, so that a conception of general contour is gained which is comparable from patient to patient. The added value of the dynamic findings, of better definition and recognition of structures by rotation of the patient or other movement, and the possibility of displaying the œsophagus by an opaque bolus far outweigh considerations to the contrary. As in physical

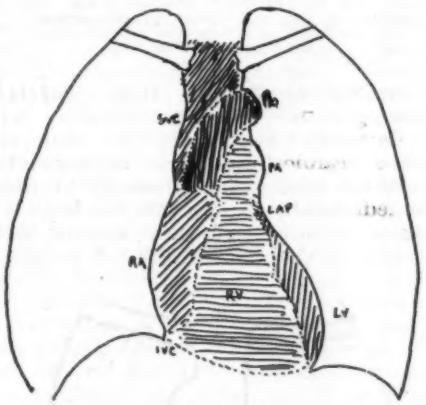


DIAGRAM I.
Normal heart, antero-posterior view (after Parkinson and Bedford). Ao: aortic knob; SVC: superior *vena cava*; PA: pulmonary artery; LAP: left auricular appendage; LV: left ventricle; RV: right ventricle; IVC: inferior *vena cava*; RA: right auricle.

diagnosis, it is essential to follow a systematic drill in regard to the details of examination. Thus the physical characters of the patient and of his bony thorax, respiratory and cardio-vascular systems, will have already been noted, and must be in the forefront of every subsequent consideration. Particular attention must be given to the presence or absence of scoliosis, kyphosis or "funnel chest". The scapulæ are separated by placing the patient's hands upon his head. Shadows of the thoracic cage and lung fields, especially the hila and bases during inspiration, the position and excursion of the diaphragmatic domes, and the root of the neck, are systematically scrutinized before any attention is directed to the cardiac outline. This part of the examination is generally facilitated by lateral and vertical shifts of the patient himself. Finally the heart is centred at a point giving the narrowest breadth of shadow at the base, and there follows meticulous examination of the cardiac contour as a whole and sectionally, in all three positions. (See Diagrams I, II and III.) Rapid canting of the patient in any plane is possible with modern apparatus and the swallowing of an opaque bolus should probably be a routine measure. Unfortunately the clinician has much less time at his disposal than the radiologist.

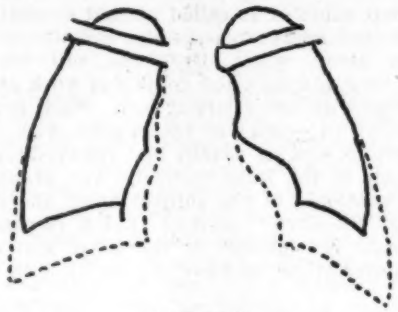


DIAGRAM 1A.
Effect of respiration upon a normal heart lying transversely and so causing a false impression of hypertrophy.

The physician's approach to any patient with potential heart disease can be summarized as follows: (i) The establishment of a diagnosis of a cardio-vascular disorder as opposed to respiratory, endocrine, constitutional or psychopathic disease. (ii) The formation of an ætiological diagnosis. (iii) A

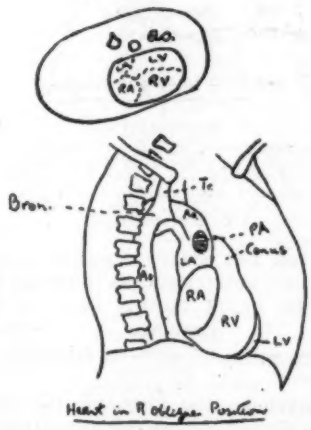


DIAGRAM II.
Normal heart in right oblique position surmounted by transverse section in mid-thoracic region. Tr: trachea; BR: right bronchus; Ao: aorta; PA: pulmonary artery; LA: left auricle; RA: right auricle; RV: right ventricle; LV: left ventricle.

consideration of the prognosis, combined with a determination of the stage reached in the natural history of the disease, and the presence or absence of complications. (iv) In a case of reexamination, to observe the results of treatment.

Screen examination can help him in the answers to all these important questions.

The Diagnosis of Heart Disease.

In very general terms the physical diagnosis in clinical medicine of a cardiac lesion depends largely upon the establishment of cardiac enlargement and upon the discovery of diastolic murmurs. From the X ray point of view the question of heart size is mainly decided from consideration of the transverse, and to a lesser extent of the longitudinal, diameter, or their product. Many years' work has been spent in the compilation of so-called normal standards for heart size and heart areas, under certain technical conditions about which Dr. Voss will tell you. Polevski[1] regards the great volume of work on these figures largely as love's labour lost. Such measurements all fail in greater or less degree, with one or two exceptions, and are usually best reserved for serial examination of the same patient. The occupation, even the pastimes, of the subject affect the norma. Smith and Bloedorn[5] showed that a cardiac area of 80 square centimetres at the lower end of the normal figures might increase in area by 38% before

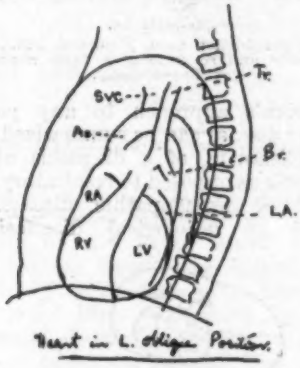


DIAGRAM III.

Normal heart, in left oblique position. SVC: superior *vena cava*; Tr: trachea; Ao: aorta; BR: right bronchus; RA: right auricle; LA: left auricle; RV: right ventricle; LV: left ventricle.

it reached even the average normal measurements (110 square centimetres) and as much as 75% before it equalled the upper normal limit of 140 square centimetres. The presence of scoliosis, high diaphragmatic level or gastric distension must be considered.

For the physician the hunt for the clinical apex in the obese female is notoriously difficult. John Parkinson[1] has said of percussion that it requires more faith than this generation possesses, and a tactile and aural sense at the disposal of few. Even during radioscopy it is hard to be sure of slight cardiac enlargement in obesity, as the organ generally lies transversely, illumination is relatively unsatisfactory, and the apex is often obscured by the diaphragm. Nevertheless one is generally somewhat further forward than before. It is necessary, however, to take care in these patients that a pad of epicardial fat at the junction of the apex with the diaphragm is not mistaken for an extension of the myocardium itself; a distinction in translucency is apparent on full inspiration. Deep inspiration often reveals a normal looking heart, which previously appeared enlarged in quiet respiration because it lay transversely. (See Diagram IA.)

The distinction between dilatation and hypertrophy is often a difficult one to decide. Hypertrophy (Diagrams IV and V) is usually revealed by specific changes in the outline of the lower left border and the apex, whereas dilatation is looked for in the more readily distensible segments of the cardiac wall.

DIAGRAM IV.

Advanced left ventricular hypertrophy—"boot-shaped heart". (Note exaggeration of cardio-vascular angle.)

In preterminal dilatation, these curves are straightened out and cardiac movement becomes feeble. Radiology has shown us that cardiac enlargement is generally regional, but not regionally general. Peter Kerley looks forward to the time when we shall be able to mix with the heart's blood an opaque substance of sufficient density to define the thickness of the cardiac wall. I do not wish,

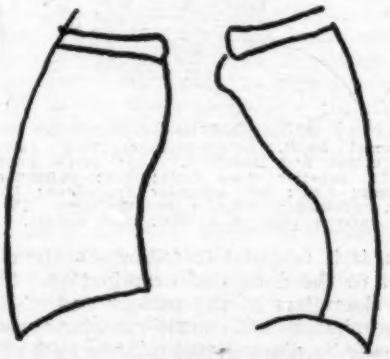


DIAGRAM V.

Right ventricular hypertrophy—"sabot-shaped heart". (Note sharp angle and vertical falling of right border to diaphragm.)

however, to over-emphasize the value of X rays in the recognition of minor degrees of cardiac enlargement; but, even in the most unfavourable circumstances, measurement of the heart by this means is preferable to the results of palpation or percussion, which may be utterly misleading or completely negative in high degrees of emphysema and obesity. The small or "drop" heart is devoid of clinical significance, though still of frequent notice

in radiological reports. The discovery of a small heart in the presence of congestive failure and venous distension should lead one to suspect the presence of Pick's disease (*concretio cordis*).

Radioscopy naturally affords unique opportunities for the differential diagnosis of cardiac affections as opposed to those of neighbouring viscera, both above and below the diaphragm, and of the mediastinum, besides revealing the cause of false displacements of the ventricular apex. Silicosis, tumours and cysts of the lungs or mediastinum, pulmonary fibrosis, diaphragmatic hernia *et cetera* are at least suspect on fluoroscopy, while the capacity to rotate the patient and to integrate or separate the extracardiac shadows from the normal cardio-vascular outline is of vast clinical assistance. Modern radiographic equipment permits of instantaneous photography in the plane of election.

Ætiological Diagnosis.

The pursuit of the cause is the watchword of modern cardiology. Radiology affords the clinician for this purpose an additional "eye", which has proved so valuable as even to supply deficiencies in the clinical history. The main diagnostic criteria of use in this respect are changes in the contour and, to a lesser degree, in the relative opacity of the individual cardiac chambers and great vessels. The chief ætiological types of organic heart disease are congenital, rheumatic, ischæmic, hypertensive, thyreotoxic, syphilitic and *cor pulmonale*.

Congenital Heart Disease.

Exact analysis of congenital heart disease is impossible during life, but right-sided positions of the heart or aorta and the four-sided syndrome of Fallot have all very definite X ray appearances. By careful correlation of the X ray appearances with auscultatory localization and the age and appearance of the patient, it is possible to determine the anatomical situation much better than ever before. Such an effort is by no means of purely academic interest; this is shown by the careful compilations of Maude Abbott and others as to prognosis. A patent *ductus arteriosus* rarely dilates sufficiently to appear in the left cardiac border, but is readily recognized clinically. It so rarely occurs alone that X ray examination generally reveals some other abnormality such as a pronounced conus bulge or right ventricular hypertrophy. Such enlargement of the pulmonary artery (Figure VIII), well seen also in the right oblique position, is a common denominator for other congenital conditions such as some auricular and ventricular defects, transposition of the great vessels, and supravalvular pulmonary stenosis, in which it reaches its greatest prominence. Pulmonary enlargement may be simulated by enlarged glands or by traction from adhesions on a normal vessel. Large auricular septum defect causes such pronounced pulmonic enlargement as to hide the aortic knob, besides causing right-sided and hilar increase. Large ventricular defects cause right ventricular hypertrophy, with retrograde right auricular enlargement and powerful systolic contraction of the right border of the cardiac silhouette. Many other anomalies have their own radiological counterpart, and assist the physician to discriminate between congenital and acquired disease, sometimes a real difficulty on purely clinical grounds. Aortic coarctation (Figure X) is generally overlooked clinically but not radioscopically. The direction and the identification of a right-sided aortic knob (Figure IX) are facilitated by an opaque bolus. In general, it helps to remember that cases of pre-natal origin are associated with greater upper pulmonary dilatation and little left auricular enlargement as compared with the acquired disease. Right ventricular hypertrophy (*cœur en sabot*) is common to both.

Rheumatic Carditis.

No evidence of value is detectable in acute endocarditis, and radiological observation is, unfortunately, of no assistance in helping the clinician to decide the duration of convalescence. One must constantly bear in mind the relative largeness of a normal child's heart, and "acute dilatation" is as bad a term to use radiologically as it is clinically. Pericarditis can be diagnosed clinically long before X ray evidence is present. Small effusions (under 400 cubic centimetres) are difficult to display. The strictly erect posture and full inspiration favour their visualization. Affected children are too ill to be examined by the screen, but bedside photography readily reveals a moderate or large effusion, the outlines of which are particularly sharp from the absence of pulsation. (Figures XII and XIII, and Diagram VI.) Serial pictures show that some fluid

DIAGRAM VI.
Pericardial effusion, right oblique position. Barium-filled œsophagus not displaced. Compare with Figure VII.

often remains after the appropriate physical signs have vanished. Radiograms taken years afterwards may reveal partial or complete calcification of the membrane. (Figure XIV.) Evidence favouring a tuberculous origin for this appearance can be searched for simultaneously. Extrapericardiac adhesions (*accretio cordis*) are detectable by indirect evidence only, such as noting the degree of cardiac sway on tilting the patient through 45°, a difficult procedure with a standard screening stand, or by undue stretching of the walls on full inspiration.

In so far as the rheumatic virus lays its hand most heavily on the mitral cusps, the discovery, on fluoroscopy, of a mitral stenosis is the key to a

rheumatic ætiology, since a short presystolic murmur may be passed over on clinical examination. On the other hand, the spectre of the systolic murmur of doubtful significance is often laid low by radioscopy. In mitral stenosis the state of affairs is revealed at once by consideration of the curve of the *conus arteriosus* and of the left auricle in the right oblique position, and this long before the patient begins to be troubled by her lesion. The mitral configuration, with miniature aorta, is recognizable even in advanced failure (loaf shape), when the left auricle may form the right cardiac border and, by pressing upwards, splays the tracheal bifurcation and presses on the recurrent nerve, and this even in the presence of distortion due to complications or involvement of other valve orifices. (Figures III, IV, V, VI, VII.) Aortic valve disease of rheumatic origin is to be suspected from high degrees of left ventricular hypertrophy, associated with a small but complete aorta, in a young person with low or normal diastolic blood pressure. Regurgitation merely adds an increase in transverse diameter to this description. The pulse pressure is a superior criterion of the grade of reflux than are radioscopic details.

Ischæmic Heart Disease.

Ischæmic heart disease means starvation or death of the whole or part of the myocardium by coronary narrowing either at the origins or during the course of the coronary vessels. The existence of coronary atheroma is usually painfully evident from the

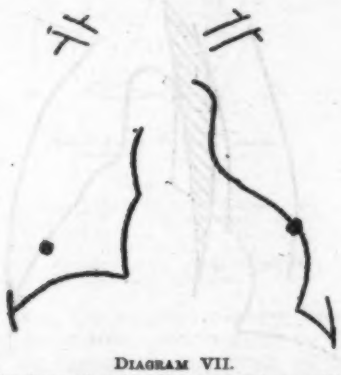


DIAGRAM VII.
Tracing of teleradiogram taken within twenty-four hours of cardiac infarction. Lead disks had been placed over the nipples. Shows dilatation of left ventricle.

clinical history, but the belief that the heart is usually normal in size in cases of *angina pectoris* is not quite true from a radioscopic standpoint. About 75% of such patients present some degree of pathological enlargement, generally associated with increased aortic capacity, tortuosity, or even visible plaques of calcification seen tangentially in the circumference of the aortic knob. (Figure XVI.) The enlargement is general, and auricular fibrillation may be present but unassociated with the extreme left atrial dilatation seen in patients with rheumatism of long standing. Additional generalized dilatation follows at once after the cataclysm of cardiac infarction. The left ventricle dilates most, according to the commonest site of the lesion. (Diagram VII.) Weeks or months afterwards a localized bulge (aneurysm of the left ventricle) may appear in this situation. (Figure XI.)

Hypertensive Heart Disease.

High degrees of left ventricular hypertrophy, combined with an increased width to the aortic arch, are the general fingerposts to a hypertensive ætiology, primary or secondary. The aorta uncoils itself and ascends towards the root of the neck (Figure II), the first part of the arch overlaps the superior *vena cava*, and the last part projects to the left of the very prominent knob seen in the antero-posterior position. (Diagram VIII.) These appearances may

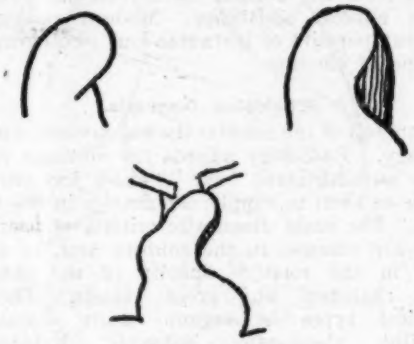


DIAGRAM VIII.
Uncoiling and kinking of the aorta as seen in hypertensive disease.

create an impression of aortic dilatation in such films, which leads to unjustified suspicions of syphilis. Signs of atheroma will coexist in the older decades. In general, the clinician will have added little here to his clinical findings from a purely diagnostic aspect.

Syphilitic Heart Disease.

Syphilitic aortic regurgitation, in my experience, is always accompanied by a radioscopically recognizable dilatation of the ascending aorta (Diagram IX),

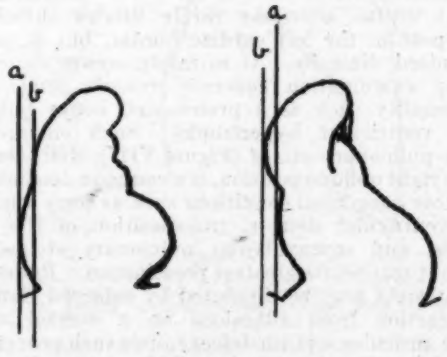
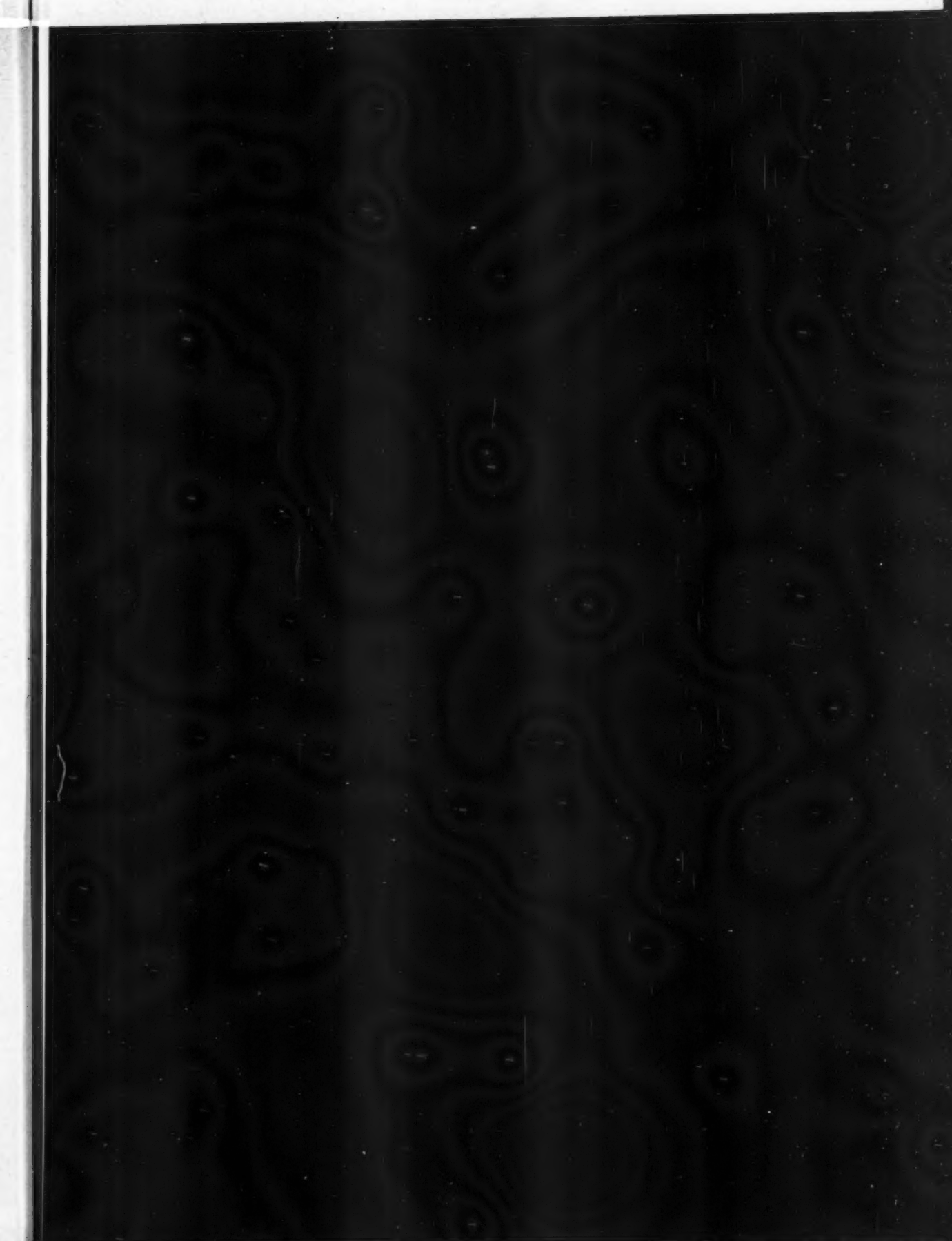


DIAGRAM IX.
Useful criterion for the diagnosis of enlargement of the root of the aorta. Tangents *a* and *b* become transposed in this condition.

and generally by an irregular fusiform enlargement of the whole arch, or even by a sacculated or resacculated aneurysm. The aorta is frequently



ILLUSTRATIONS TO THE ARTICLES BY DR. KEMPSON MADDOX AND DR. KERROD B. VOSS.

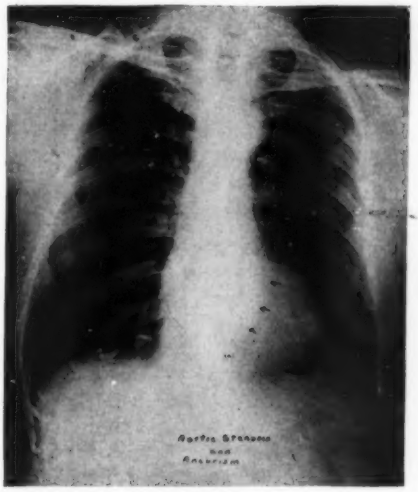


FIGURE I.

Aortic stenosis, with aneurysm of the descending aorta. The aorta is lengthened and slightly dilated in the ascending part. There is slight enlargement of the left ventricle.

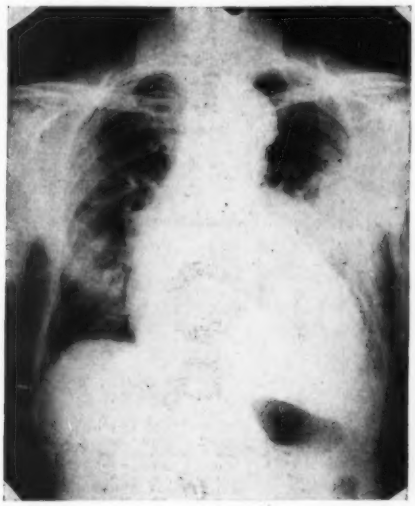


FIGURE II.

Aortic incompetence. The aorta is dilated, especially at its origin, and also shows atheromatous plaques. The left ventricle is greatly enlarged; the left border of the heart meets the diaphragm at right angles, indicating some dilatation as well as hypertrophy. There is engorgement of the pulmonary vessels with a pleural effusion on the left side.

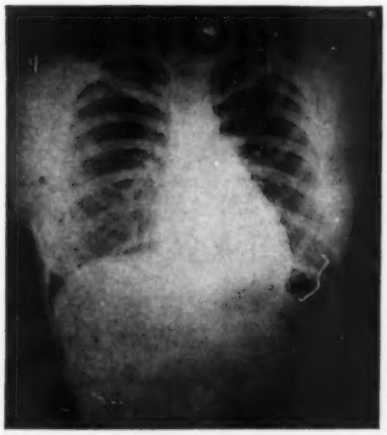


FIGURE III.

Mitral stenosis. The heart is of the vertical type and there is enlargement of the curves of the *conus arteriosus* and left auricle, causing obliteration of the cardio-vascular angle.

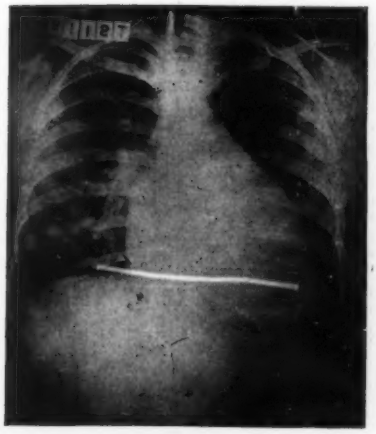


FIGURE IV.

Mitral incompetence. The changes are more pronounced than in Figure III. All the chambers of the heart are enlarged and there is some engorgement of the pulmonary vessels. The cardio-vascular angle is partially restored owing to enlargement of the left ventricle.

ILLUSTRATIONS TO THE ARTICLES BY DR. KEMPSON MADDOX AND DR. KERROD B. VOSS.

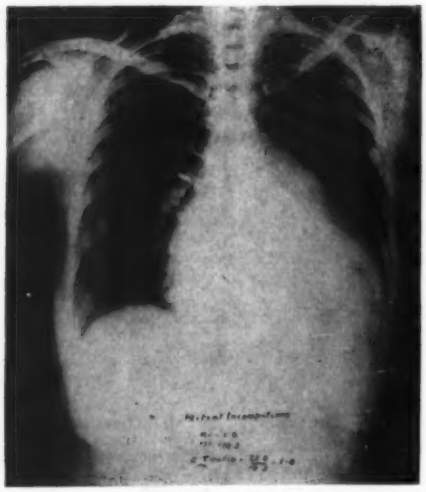


FIGURE V.

Mitral incompetence. A more advanced stage. The generalized enlargement is much more pronounced and the cardio-vascular angle is replaced by a convex curve, due to the enlarged right ventricle pushing across the dilated conus and left auricle. The œsophagus was displaced posteriorly and to the right.

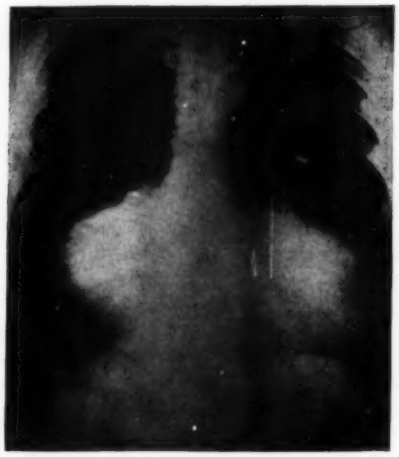


FIGURE VI.

Mitral incompetence. A very advanced stage. The enlarged left auricle projects a long way past the right border of the heart, giving the heart a symmetrical appearance like a calabash.

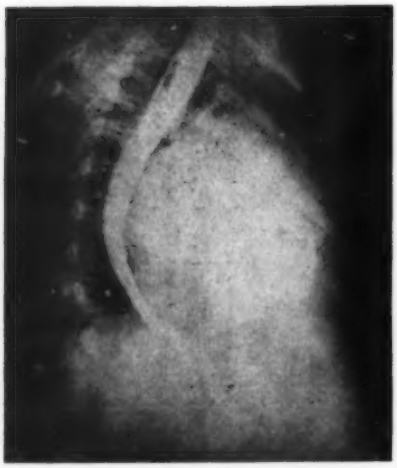


FIGURE VII.

Mitral incompetence. An oblique view of the heart shown in Figure VI, showing the displacement of the barium-filled œsophagus.

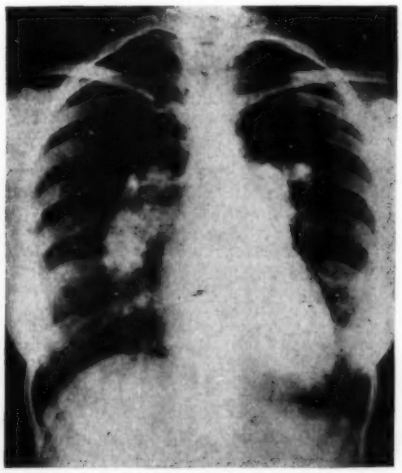


FIGURE VIII.

Congenital heart disease. Accentuation of the curve of the pulmonary artery, probably due to congenital pulmonary stenosis and/or a patent *ductus arteriosus*. Note the "gas-pipe" appearance of the right pulmonary artery.

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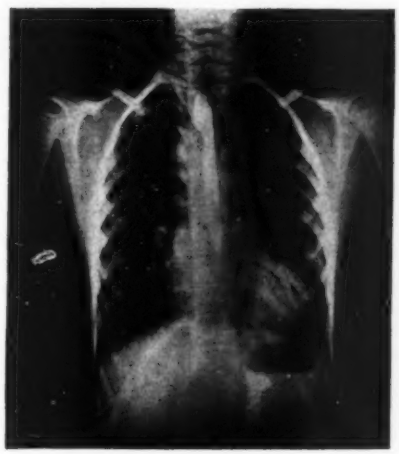

FIGURE IX.

Situs inversus aortæ. The indentation in the right border of the barium-filled œsophagus is clearly shown.

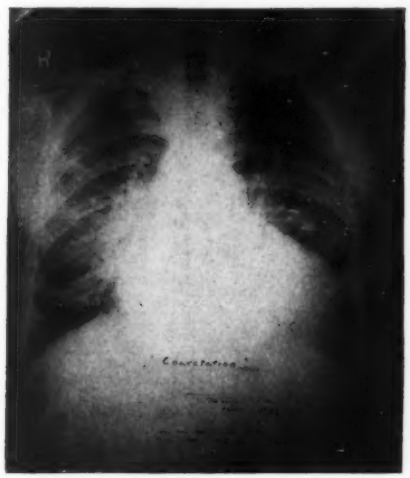

FIGURE X.

Coarctation of the aorta. An excellent example, showing four of the signs. The actual stenosis could be seen in the oblique view. (By courtesy of Dr. O. A. Diethelm and Dr. B. P. Anderson Stuart.)

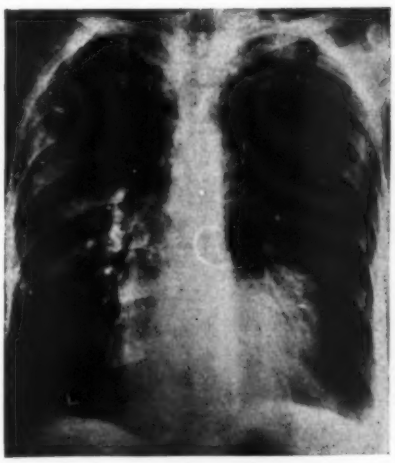

FIGURE XI.

Aneurysm of the wall of the left ventricle following cardiac infarction.

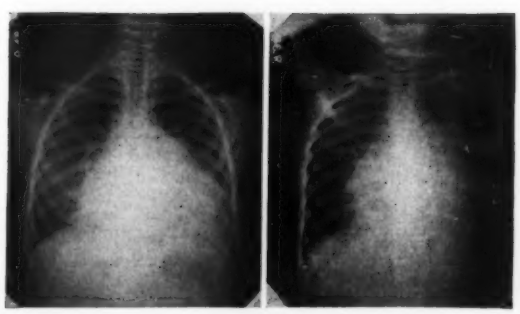

FIGURE XII.

Pericardial effusion in a child. The view on the left was taken with the child erect and shows the heart sagging towards the diaphragm. The view on the right was taken with the child prone and shows how the heart tends to adopt a more globular configuration in that position, with upward extension of the shadow.

ILLUSTRATIONS TO THE ARTICLES BY DR. KEMPSON MADDOX AND DR. KERROD B. VOSS.

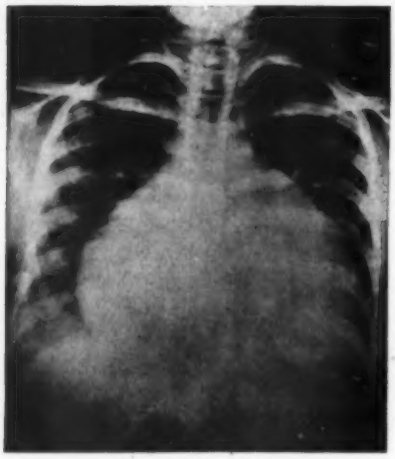

FIGURE XIII.

Pericardial effusion. A large effusion in an adult causing a globular appearance.

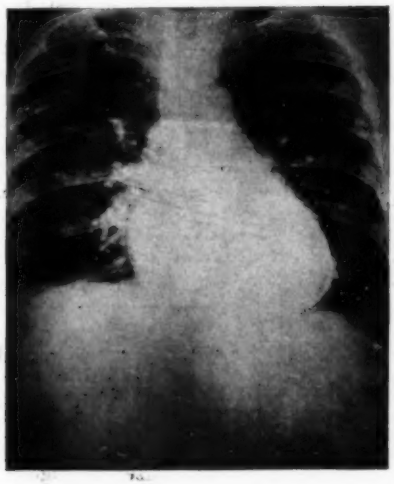

FIGURE XIV.

Pericardial calcification. The calcified plaques can be seen along the left border of the heart.

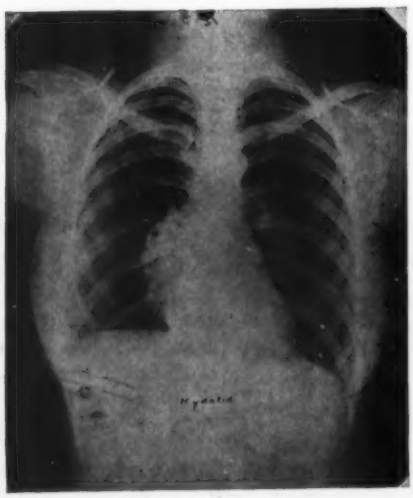

FIGURE XV.

Calcified hydatid cyst of the heart wall. This may have commenced in the pericardium and invaded the heart wall. The patient had no cardiac symptoms and this was found during a radiographic examination of the gastro-intestinal tract. The characteristic calcification is well shown.

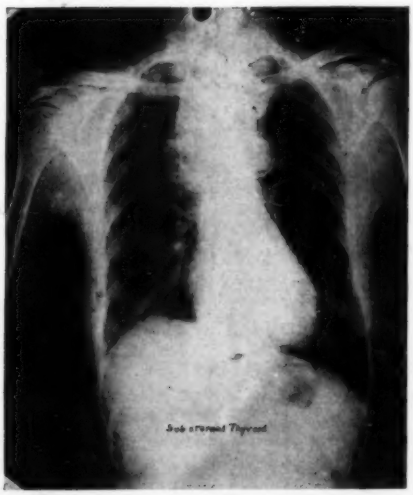

FIGURE XVI.

Substernal thyreoid. There is considerable enlargement of the thyreoid, which is displacing the trachea to the right. Areas of calcification can be seen in the thyreoid both above and below the level of the clavicles, and there are also to be seen well-marked calcified atheromatous plaques in the wall of the aortic bulb.

irregularly opaque. Difficulties of differential diagnosis of aneurysms are usually readily overcome under satisfactory technical conditions of screening. (Figure XV.) It is surprising to the clinician to see how frequently the innominate artery shares in these lesions.

Thyreotoxic Heart Disease.

I have been led on more than one occasion to suspect the presence of hyperthyreoidism, by rapid dancing or tugging movements of the heart in a vertical plane, visible in many of these patients. It is always difficult for the physician to answer the surgeon's query as to the state of the myocardium in this disease. Many of his clinical criteria, and all functional tests, are valueless, while the cardiogram is not very helpful. Fortunately the disease most usually appears before the atheroma age, and, since little else but operation will cure the fibrillation and the patient, it is unwise to raise the bogy of a cardiac death, unless very definite enlargement of the heart shadow is apparent. Minor degrees of generalized dilatation are common, but exact measurements should be made, as a very rapidly acting heart often gives a false impression of increased size. The discovery of the flask-shaped shadow of a retro-sternal goitre, moving on deglutition, may give the clue to the cause of an obscure paroxysmal fibrillation. In such patients this area should, therefore, be diligently scrutinized. (Figure XVI.)

Heart Failure Secondary to Pulmonary Disease (Cor Pulmonale).

The presence of emphysema in all conditions in which coughing is continued for long periods renders clinical examination of the heart most difficult. Hypertrophy of the right ventricle (Diagram V) in acquired pulmonary obstruction is readily visible as a sharp angulation of the left border with a vertical descent to the diaphragm. This is due to rotation of the heart, whereby the right ventricle replaces the left in the formation of the lower left edge of the shadow. (Diagram X.) The left oblique position confirms this enlargement, and allows of reobservation of the conus, which also bulges convexly. The condition of the pulmonary artery and its branches, its encroachment upon the subaortic window in the right oblique position, the definition of the right pulmonary artery between right bronchus and lung, the state of the lung markings, the exact extent of the causative pulmonary lesion and the efficiency of the tricuspid valve are all informative to any experienced observer of these morbid states.

Progress, Prognosis and the Effect of Treatment.

Prognosis is difficult above all in myocardial disease. It demands a logical consideration of every aspect, social, familial, clinical and instrumental. Radiology offers help in the following respects: the more exact determination of the stage reached in the lesion; the presence of additional deteriorative factors, such as atheroma; the presence of pulmonary complications, either primary or secondary, to the cardiac damage itself; and the arrest or subsidence of certain morbid appearances either spontaneously or following specific treatment.

Examples of the kind of assistance that may be given in various types of heart disease are as follows:

(*a*) *Rheumatic Carditis.*—The exact degree of dilatation of the left auricle; the confirmation of the supersedence of pure mitral stenosis by mitral incompetence (Figure V); the degree of congestion of the pulmonary vascular system and great veins; the detection of the ovoid shadow of pulmonary infarction; the advent of tricuspid incompetence; and the first signs of fluid in the right pleural sac, besides the rate of disappearance of a pericardial effusion.

(*b*) *Hypertensive Heart Disease.*—The state of the lung fields as evidence of left-sided myocardial integrity or failure; the degree of associated

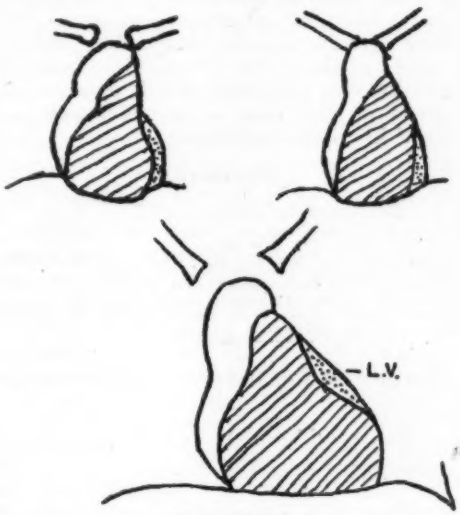

DIAGRAM X.

After Polevski. Designed to show progressive encroachment of the right ventricle on the left border of the heart in any case of pulmonary obstruction.

atheroma, the condition of the right auricle; subsidence of purely dilatory changes after rest and associated therapy, at which time the paradox of redarkening of the lung fields may be seen as the result of better output from the right ventricle.

(*c*) *Syphilitic Heart Disease.*—The rate of increase in aortic dilatation and its arrest or otherwise following specific therapy, in addition to the signs noted above under (*b*).

(*d*) *In Thyreotoxic, Post-Pneumonic, Post-Typhoidal Myocarditis et cetera.*—The rate of return to normal of right-sided or general increase in breadth of the cardiac silhouette.

Conclusion.

Enough has probably been said to indicate the great value of routine radioscopy in clinical cardiology, and it is hoped that before long some satisfactory form of bedside screening will be available for examination of sick patients. It may even appear that in many cases no other form of inquiry is necessary. In certain instances, such as in mitral

stenosis, aortitis, left ventricular hypertrophy, we may as well admit this at once. Some of our clinical methods have long been obsolete, but are rightly retained for training purposes or for situations in which X ray facilities are unavailable. On the other hand, it often happens that the radioscopic image is less informative than the eyes, hands and stethoscope, for example, in early regurgitant murmurs, some forms of congenital disease and disorders of rhythm. In *angina pectoris* screen examinations may occasionally yield no more information than the physical signs. Again, fallacies in X ray diagnosis are naturally common enough, but from his unique position the clinician is less likely to be wrong than any one else, as he often has only to look for confirmation of what he has already guessed is present. By doing so, however, he makes his diagnosis doubly sure and can then prognose and treat with a confidence conscientiously impossible before the development of the X ray screen, while from time to time he discovers something which puts an entirely new aspect on the case or even demands a total revision of his clinical opinion.

References.

(1) John Parkinson: "The Radiology of Heart Disease", *The British Medical Journal*, September 30, 1933, page 591.
(2) Peter Kerley: "Radiology in Heart Disease", *The British Medical Journal*, September 30, 1933, page 594.
(3) Crighton Bramwell: "Radiological Diagnosis of Cardiac Enlargement", *The British Medical Journal*, September 30, 1933, page 597.
(4) J. Polevski: "The Heart Visible", 1934, page 54.
(5) H. W. Smith and W. A. Bloedorn: "The Size of the Normal Heart: A Teleroentgen Study", *United States Medical Bulletin*, 1922, Volume XVI, page 219.

RADIOLOGY IN RELATION TO HEART DISEASE.[1]

By KERROD B. VOSS,
Sydney.

THE object of a radiological examination of the heart is to determine its size, shape and position and the nature of the pulsations.

There are four methods of examination: fluoroscopy, orthodiagraphy, teleroentgenography, kymography. By using one or more of these methods we obtain radiological information as to the size, shape, position and pulsations of the heart, and whether any or all of these present any variation from the normal; but before we can state whether any abnormality is present, we must be thoroughly acquainted with the normal.

In different persons the normal heart varies considerably in size and shape, and also in position. In the asthenic type the heart tends to be slender and central. This type of heart is so often found in association with pulmonary tuberculosis that it is commonly referred to by various authors as a "tuberculous heart", but it is consistent with perfect health.

In the sthenic type the heart tends to be more transverse, and in obesity, owing to the accumulation of intraabdominal fat and consequent elevation of the diaphragm, the heart lies very transversely, with the apex well out towards the axilla.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 28, 1936.

At birth the heart is much larger in relation to the thoracic cage and lungs than it is in adult life, and one must be very chary of reporting an enlarged heart in a young infant. Inspection of many normals must be made before one can feel at all fitted to report on an infant's heart. During the first year of life the heart increases very little in size, whereas the lungs increase in volume sixfold, so that by the end of this first year the cardio-thoracic ratio is almost equal to that of the adult.

I shall pass now to a more detailed description and discussion of the various methods of examination, with their advantages and disadvantages.

FLUOROSCOPY.

Fluoroscopy is perhaps the most important method of all, and certainly the most convenient and cheapest. By it we can examine the heart from all aspects and see the actual pulsations. On many occasions an unusual shadow may be seen near the heart, and fluoroscopy is generally the best means of determining whether it is cardiac, vascular, pulmonary or mediastinal in origin. The chief disadvantage of the method is that with an ordinary screening stand distortion is at a maximum owing to the tube being so close, but this distortion is reduced in special heart-screening stands by being able to move the tube back to a distance of six to seven feet from the screen. With the tube well back, there is greater danger to the observer by rays streaming past the patient, and great care has to be observed in keeping the beam as small as possible.

In spite of the distortion, this is a most valuable method. From it one can ascertain the silhouette and so the type of heart, and after the study of many normals—distorted normals—one can say with a considerable degree of certainty whether any enlargement is present.

During fluoroscopy it is often advisable to give the patient an opaque bolus to determine whether there is any displacement or compression of the œsophagus, such as occurs, typically, with the enlarged left auricle found in mitral stenosis.

ORTHODIAGRAPHY.

Orthodiagraphy is an extension of the first method and is an attempt to record graphically what has been seen, by drawing in the outline of the heart on a piece of glass, film or clear paper attached to the front of the screen. It has the same advantages as fluoroscopy, namely cheapness and convenience. The distortion is reduced to a minimum by using only the smallest possible beam of light and moving this beam round the cardiac outline, drawing as one goes. Theoretically the distance of the tube from the screen should not matter if the beam is small, but as an added safeguard against distortion the tube may be moved back from six to seven feet. During the process of drawing in the outline the state of respiration must be observed and care must also be taken to map only the diastolic outline. In addition, in order to eliminate parallax, the beam must be very carefully followed round with the eyes, the direction of vision always being kept perpendicular to the screen at the point being mapped. As one

observer put it, a pencil attached to the tip of the nose would be the ideal.

In spite of all these precautions, errors are apt to creep in, and an error of ±1 to 2 centimetres in the transverse diameter may occur even with a skilled observer. The chief advantage of this method is that it furnishes a cheap and permanent record that can be filed with the patient's papers.

TELEROENTGENOGRAPHY.

Teleroentgenography consists in taking skiagrams at a sufficient distance to minimize distortion, thus providing a permanent record that can be studied and measured at leisure. The distance at which the skiagrams should be taken has occasioned much discussion. Most chest stands are built to allow of skiagrams being taken at six feet, but for cardiography some advocate seven feet. In Europe much of the work is done at two metres (six feet six and three-quarters inches), but some think it should be two and a half or three metres (nine feet ten and one-eighth inches). In Germany an attempt was made at forty feet, but the cost in tubes at this distance would be great. The ideal distance is infinity, but this is impracticable, and we must restrict ourselves to a more mundane distance. It does not really matter much whether we use six feet or seven feet, two metres or three metres, providing we always use the same distance, as, short of infinity, there must be some distortion; and by always using the same distance, we adapt ourselves to the distortion obtained at that distance. Simple calculation will show that for a point, say, eleven centimetres from the mid-line and eight centimetres from the film, the distortion is five millimetres at six feet and 4·3 millimetres at seven feet; in other words, the increased distortion at six feet is only 0·7 millimetre, which is well within the margin of error. At two metres the distortion is 4·5 millimetres, and at three metres it is three millimetres, an increase of 1·5 millimetres, which is again scarcely appreciable.

In an orthodiagram the error is variable and may be ±1 to 2 centimetres, or even more. In a teleroentgenogram the error is a constant one owing to distortion, and is always positive, with a maximum of about five millimetres, and may thus be allowed for by subtraction, whereas in an orthodiagram the error may be + or −.

The positions commonly used are postero-anterior, first or right-oblique and second or left-oblique, with the patient standing erect, and postero-anterior with the patient lying down.

There are two methods of studying the skiagrams obtained, the qualitative and the quantitative.

The Quantitative Method.

The quantitative method consists in measuring the distance between certain landmarks on the heart borders and comparing these measurements with those of normal hearts. This can be, at best, only an approximate method, and is an attempt to convey numerically the degree of enlargement of the heart as a whole, and of its various chambers. Numerous authors have proposed methods of making the measurements and have supplied sets of figures purporting to be those of normal hearts. Probably the first method proposed was the use of the cardio-thoracic ratio, which is the ratio of the transverse diameter of the chest to the transverse diameter of the heart. This method is favoured by several German workers (Hammer, Zondek, Groedel and Assmann), and the ratio has an average value of two.

Moritz took three diameters, the longitudinal, transverse (composed of the sum of the right and left median distances) and the basal, and correlated these with the patient's height and age. Claytor and Merril used the same diameters and correlated them with weight. Dietlen also used the same diameters and plotted them against height, weight and age, with the patient erect or supine. Vaquez and Bordet used these diameters and took as their comparative factors the patient's weight and age. They later used a more complex system of measurement in an attempt to determine the size of the individual chambers, but, as a general rule, they appear to rely on the longitudinal and transverse diameters and the cardio-thoracic ratio.

O'Kane, Andrews and Warren, and later Fray, studied the heart in the left-oblique position with an angle of rotation of about 45°. The main objection to the plain postero-anterior view is that it gives no idea of the depth of the heart, and theirs is an attempt to remove this disadvantage. In the left-oblique position the inter-ventricular septum is approximately in a plane perpendicular to that of the film, and Fray has devised a method of plotting on the skiagram the position of the septum, so that the size of each ventricle can be measured separately. He compares the oblique diameter of the heart obtained in this way with the oblique diameter of the right half of the thorax and obtains a ratio of 2·02 for the average, which is very close to that for the postero-anterior cardio-thoracic ratio.

The area of the heart in the postero-anterior view has been measured, and various values, ranging from 98 to 131 square centimetres, have been proposed as normal. Hodges and Eyster devised a most ingenious formula for the normal area. It is very complex.

$$\begin{aligned}\text{Normal area} = {} & \text{Age (in years)} \times 0{\cdot}0204. \\ & + \text{Height (in centimetres)} \times 0{\cdot}8668. \\ & + \text{Weight (in kilogrammes)} \times 0{\cdot}337. \\ & - 63{\cdot}8049. \\ & + 1 \text{ (if age exceeds 45 years).}\end{aligned}$$

The Qualitative Method.

In the qualitative method the shape of the heart is studied in several planes and, from the shape, a determination is made whether one or more chambers are enlarged, without an attempt to give a numerical index of the degree of enlargement.

Lesions Producing Characteristic Appearances.

Most of the lesions occurring in the heart, by producing enlargement of certain chambers, give rise to very characteristic shapes or silhouettes of the heart, which are readily recognized, for example the aortic, mitral, pulmonary, tricuspid and congenital types, the myocardial degeneration type and pericardial effusion. The salient features of these are as follow:

The Aortic Type.

The occurrence of the aortic type does not necessarily indicate an aortic lesion, but merely increased blood pressure.

Aortic Stenosis.—In aortic stenosis (Figure I) the left median distance is not greatly increased. The left lower border is rounded, but may meet the diaphragm approximately at right angles, indicating the presence of some dilatation. The aorta may or may not be dilated. The cardio-vascular angle is well marked.

Aortic Incompetence.—In aortic incompetence (Figure II) the left median distance is greatly increased. The left lower border moves out and tends to meet the diaphragm at right angles. The aorta is dilated. The cardio-vascular angle is accentuated. The heart is shaped like a duck.

Mitral Type.

Mitral Stenosis.—In mitral stenosis (Figure III) the heart is of the erect type and tends to be narrow. The pulmonary artery and left auricle are enlarged and obliterate the cardio-vascular angle. The heart rotates and the aortic knob becomes less prominent. The hilar markings are increased. The left auricle encroaches on the retro-cardiac space, and may project beyond the right border of the heart. The œsophagus is compressed and displaced and interference with deglutition may take place. The angle of bifurcation of the trachea is increased (the average is 40° to 70°, the mean 58°; in mitral stenosis the average is 78° to 117°).

Mitral Incompetence.—In mitral incompetence (Figures IV to VII) the heart still maintains a vertical tendency. The left median distance is increased. The cardio-vascular angle is at first partially restored as the left ventricle enlarges, but is lost again as the right ventricle enlarges and pushes the *conus arteriosus* and pulmonary artery across. In the left-oblique view the heart has a globular appearance owing to the fact that all the chambers are enlarged. The œsophagus is invariably displaced.

Combined Mitral and Aortic Lesion.

In a combined mitral and aortic lesion the features of both types are combined and the left border of the heart has a pronounced step-ladder appearance.

Pulmonary Type.

Pulmonary Stenosis.—Pulmonary stenosis is generally congenital.

Pulmonary Incompetence.—In pulmonary incompetence the right side of the heart is enlarged, but the outstanding feature is a well-marked pulsation of the pulmonary vessels, to be seen on fluoroscopy.

Tricuspid Type.

Tricuspid Stenosis.—Tricuspid stenosis is generally congenital.

Tricuspid Incompetence.—In tricuspid stenosis the right side of the heart is enlarged and the superior *vena cava* is prominent.

Congenital Lesions.

Numerous types of congenital defects can occur in the heart. Most of them produce more or less characteristic changes in the appearance of the heart, but several defects may be present at the same time, thus tending to obscure the typical picture. The more common lesions are as follow:

Pulmonary Stenosis.—Pulmonary stenosis results in a widening of the pulmonary artery which is more pronounced when the stenosis is near the bifurcation. The œsophagus is not displaced, since the left auricle is not enlarged; this serves to distinguish it from the mitral type of heart, with which it may be confused. (Figure VIII.)

Patent Ductus Arteriosus.—A patent *ductus arteriosus* also causes enlargement of the pulmonary curve, but is a less common cause of enlargement than is pulmonary stenosis. The two conditions cannot be distinguished with any certainty radiographically, and the differentiation must be made clinically.

Auricular Septum Defect.—In the presence of an auricular septum defect the pulmonary curve is much enlarged and the cardio-vascular angle is diminished or obliterated. The aorta is narrow. The right median distance is increased and occasionally the left. The hilar shadows are increased in size and the lungs are darker than normal.

Ventricular Septum Defect.—In the presence of a ventricular septum defect the right median distance is increased owing to enlargement of the right ventricle and possibly of the right auricle; the left median distance may also be increased, but to a less degree, giving a symmetrical-looking heart. The pulmonary curve may be accentuated.

Transposition of the Great Vessels.—Transposition of the great vessels is incompatible with life unless there is an associated septum defect.

Tricuspid Incompetence.—In tricuspid incompetence the pulmonary curve is accentuated. The right ventricle is enlarged, and may form part of the left border of the heart. The lung fields are not dark.

Congenital Sclerosis of the Pulmonary Vessels.—Congenital sclerosis of the pulmonary vessels is a form of *endarteritis obliterans*. The pulmonary curve is very enlarged and the lung fields are unusually bright.

Dextrocardia.—Dextrocardia usually, but not invariably, occurs in association with a complete transposition of viscera. The condition is not uncommon. It may be missed if only skiagrams are taken, but is self-evident on fluoroscopy.

Situs Inversus Aortæ.—In *situs inversus aortæ* the aortic bulb is seen projecting to the right and an opaque bolus shows the œsophagus to the left with its right border indented by the aorta. (Figure IX.)

Coarctation of the Aorta.

Coarctation of the aorta presents very characteristic and striking features (Figure X), namely: (i) The aortic knob is absent. (ii) Enlargement of the left ventricle generally occurs. (iii) The ascending aorta is dilated. (iv) The under-surface of the ribs is

notched and eroded owing to the enlarged intercostal vessels. (v) There is a defect in the descending limb just below the origin of the subclavian artery; it is difficult to demonstrate owing to the presence of the spine.

Myocardial Degeneration.

In myocardial degeneration the heart tends to lie transversely, but has a very squat appearance. The left border is very straight. The heart may get smaller on deep inspiration.

Coronary Sclerosis.

Sosman and Washika have reported three cases of coronary sclerosis in which they demonstrated calcification in the coronary arteries.

Calcification of Valves.

Calcification of the valves can be demonstrated in good skiagrams when it is present.

Aneurysm of the Heart Wall.

Aneurysm of the heart wall is not common, but has a typical appearance (Figure XI); it may occur as a consequence of coronary occlusion.

Pericardial Effusion.

When pericardial effusion is present and the patient is in the erect position, the heart has a rather triangular appearance and the borders may be straight, as in myocardial degeneration, with obliteration of the cardio-vascular angle. When the patient is in the prone position, the heart assumes a more rounded appearance, the borders are more convex and the shadow towards the base becomes wider (Figures XII and XIII.)

Pericardial Calcification.

The plaques of pericardial calcification are well shown in the skiagram when present. (Figure XIV.)

Hydatid Cyst.

The characteristic appearance of a calcified hydatid cyst cannot be mistaken in this country. (Figure XV.)

KYMOGRAPHY.

Kymography is the latest method of examination, but it has not yet achieved widespread use. The principle of the method consists in placing a grid with numerous fine, regularly-spaced slits between the patient and the film. During the exposure, which occupies a time at least equal to one complete normal cardiac cycle (that is, about one-seventieth of a minute), the film moves downward at a uniform speed for a distance slightly less than that between the slits. The resulting kymogram shows the position at successive instants of numerous equidistant points on the heart border, and represents in wave form the movement of these points during the cardiac cycle.

Uniformity of opinion has not yet been reached as to the best values for width and spacing of slits, and hence for velocity of film. A fine slit and a fairly high speed of film would give excellent detail, but the resulting equivalent exposure presents great technical difficulties at the present time, and a compromise must be made. The factors proposed by a few of the leading workers are set out in the accompanying table.

Observer.	Slit Width in Millimetres.	Velocity in Millimetres per Second.	Total Exposure in Seconds.	Equivalent Single Exposure in Seconds.
Goeth	3·0	17	3·5	1/8
Cigolini	3·0	50	1·0	1/10
Stumpf	0·5	4	3·0	1/8
Hirsch	0·4	12	1·0	1/30

Stumpf studies his kymograms by means of what he calls densograms. These are formed by passing a fine pencil of light through the film and moving the film in the same direction that it moved during the exposure.

As the density of the heart varies during a cycle, so the density of the kymogram varies, but in the inverse ratio. By a suitable arrangement of photo-electric cell and reflecting galvanometer a graphic record of any desired magnification can be obtained of the varying density of the heart in each zone, and this record represents accurately in wave form the movements of the heart, just as the kymogram does. By studying the kymogram or densogram, one can determine the exact nature of the movements at numerous points on the heart border, and the time-relationship of the movements at these points. If a shadow is seen, apparently continuous with the cardiac area, it is possible, by studying its pulsations, to determine whether it is auricular, ventricular or vascular in origin, or whether its pulsations are transmitted and not expansile, in other words that it is of extra-cardio-vascular origin.

Kymograms have been made with a recording stethoscope linked in parallel with the kymographic apparatus. By this means it was possible to determine at exactly which points of the cardiac cycle the various heart sounds occurred. Similarly, kymograms have been made coupled with an electrocardiograph.

Kymography is still in its infancy, but it will undoubtedly and in the near future become of great importance in the radiological study of the heart.

Reports of Cases.

CHINK ANTRUM: A REPORT AND CONSIDERATION OF A RARE ANATOMICAL CONDITION.

By CECIL CANTOR, M.D., B.S. (Melbourne), F.R.C.S. (Edinburgh), D.L.O. (London), F.R.A.C.S. (L.O.),
Melbourne.

CHINK ANTRUM is of interest both to anatomists and otologists, and its possibility must be kept in mind by surgeons undertaking any type of mastoid operation. Its occurrence renders the operation exceedingly difficult and greatly increases the danger of damage to the facial nerve.

Miss A.C., aged twenty years, was seen in the aural department at the Alfred Hospital on June 6, 1933. She had a left-sided otorrhœa of three years' duration and had

previously attended on several occasions. She was now complaining of an increase in the amount of discharge and pain in the ear.

On examination the left meatus was found to be full of pus. This was cleared away, but, owing to the extreme narrowness of her meatus, it was impossible to examine the tympanic membrane. Within a few minutes her meatus was full of pus again.

There was considerable tenderness over the mastoid process, at the tip, over the suprameatal triangle (which is more definite evidence of mastoid infection), and along the posterior margin of the mastoid (Griesinger's sign). Griesinger's sign is supposed to be evidence of infection of cells at a distance from the antrum and is regarded as a sign for operation. Her hearing was bad. Accurate tests were not undertaken.

The right external meatus was similarly very narrow, and it was again impossible to examine the drum.

She was admitted to the ward, and on June 9, 1933, a left mastoid operation was undertaken, the exact nature to be determined by the pathological findings. The cortex of the mastoid process was completely acellular and the bone extremely difficult to remove, being of the eburnated type. After the usual procedure no evidence of the antrum was found, but the dura of the middle cerebral fossa was exposed. This dipped down below the upper level of the meatus, encroaching on the site usually occupied by the mastoid antrum. The lateral sinus was not exposed and was not, contrary to expectation, far forward. No trace of antrum could be found posteriorly. The cartilaginous meatus was then dislocated and a fine bent probe was placed in the aditus from in front. Instead of passing into the antrum it ended blindly in a tiny chink at the posterior end of the aditus. By resection of the outer attic wall and by lowering the posterior meatal wall this was exposed.

The accepted teaching is that a mastoid antrum is always present. This tiny blind end was presumed to be an antrum of the very rare "chink" type, and was undoubtedly the site the mastoid antrum would have occupied in its normal condition had pneumatization occurred. The aditus at this point was 4·375 centimetres (one and three-quarter inches) from the surface. It is stated by most authorities that an antrum is always present. Thus Gray states that the tympanic antrum is a cavity of considerable size at the time of birth. J. S. Frazer states that the tympanic antrum is always present from birth onwards. According to F. W. Watkyn-Thomas, the antrum is always present, although in rare cases it is a minute chink behind the aditus. Watkyn-Thomas had seen one such case in fifteen years. Paradoxically this author states earlier in his book: "The mastoid antrum is practically always present."

The explanation of the condition described above is one of arrested development. Early, the mastoid antrum and mastoid air cells develop from the tubo-tympanic pouch. The middle ear develops, according to Guthrie, at the expense of the embryonic gelatinous tissue which at first fills the greater part of the middle ear cleft, that is, the space bounded by the cartilaginous and bony walls of the Eustachian tube, tympanum and antrum. Towards term the walls of the middle ear cleft are wholly freed of gelatinous tissue and become fully lined with mucous membrane, which extends throughout the Eustachian tube, tympanum and antrum. Hence the mastoid antrum is present from birth onwards, but it is later on that the mastoid air cells begin to appear.

Pneumatization is almost complete about the age of five. It may still continue after this, but the process is much less active. If practically complete pneumatization has not occurred when the child is five years old, the condition is one of arrested development.

It is well known that extensive pneumatization does not occur in every temporal bone. Cheatle showed that in 80% of cases it does occur. In the remainder it fails in varying degrees, the complete failure giving the dense acellular bone.

The causation of departure from the normal (if complete pneumatization is regarded as normal) is not definitely known. Until recent years it was held that the eburnated sclerotic type of mastoid was the result of long-continued suppuration, producing a condition of osteosclerosis, with obliteration of the air cells. According to Wittmaack, of Hamburg, the sclerotic mastoid is the result of prenatal or infantile inflammation, "a hyperplastic infantile *otitis media*".

The vitality of the mucous membrane is damaged because of the infection, and normal pneumatization does not occur. It can be shown, however, that skiagrams of mastoids of children who have had infantile inflammation or even had mastoid operations performed, show normal pneumatization some years afterwards.

Cheatle's views are now generally accepted, and pneumatization of the mastoid depends on the type of bone and on the activity of the mucosa. Hence the acellular sclerosed type is one in which development has ceased, probably because of the inherent nature of the bone and not because of inflammation.

This explains the depth from the surface of the rudimentary antrum. Although it is part of the early middle ear cleft, and although pneumatization commences from the antrum, it is probable that because of the nature of the bone there was arrested development of the antrum and non-pneumatization of the mastoid. Had the antrum been of the normal type, pneumatization, either partial or complete, would have taken place and it would have approached to the normal depth of not more than two centimetres from the surface. It also explains the extreme narrowing of the external meatus. With pneumatization and absorption of bone from the mastoid process widening of the meatus would occur.

In view of the narrowing of the meatus on the opposite side it is probable that a similar condition existed there. There are many other views as to non-pneumatization.

Mouret states that non-development of the air cells is due to some individual influence, "a personal tendency". In other words, the eburnated mastoid is a type, though rare, just as the cellular mastoid is a type. Alexander and Brock state that it is due to insufficient aeration of the middle ear cleft caused by adenoids. According to Albricht, the degree of pneumatization depends upon the energy of the epithelium and the resistance offered to it by the surrounding tissues. The function of the epithelium is to proliferate and follow the regressions of the subepithelial connective tissue as this extends into the preformed spaces in the bone.

Wittmaack's theory of hyperplastic otitis as a cause of non-pneumatization has as its basis the inability of the epithelium to proliferate. There is not only a hyperplasia of the epithelium, but of the subepithelial connective tissue as well. It does not seem probable that hyperplasia of epithelial tissue alone will prevent osteoclastic resorption of bone.

A case was described by Marx in which extensive pneumatization occurred along with hyperplastic changes in the epithelium.

The type of mastoid described in this paper is a danger to both the patient and to the surgeon. The density of the outer wall prevents escape outwards and masks signs of inflammation. There is always a greater risk of penetration of infection through the comparatively thin walls of the tegmen into the middle and posterior cerebral fossæ, with consequent serious cerebral complications. This patient ultimately died of a large chronic temporosphenoidal abscess.

To the surgeon there is the greater risk of damage to the facial nerve, as, with continued excavation and inability to find the mastoid antrum, he approaches nearer to its course. In practically all cases of simple mastoid operation in which this catastrophe does occur there has been difficulty in finding the mastoid antrum.

It is advisable, therefore, if one is unable to find the antrum, after a careful search on anatomical lines, in or about its usual site, to approach the aditus from in front, via the tympanum. Using this as a guide, with or without resection of the outer attic wall, expose the aditus and antrum by removing the overlying bone. This constitutes practically a reversed attico-antrostomy or epitympano-mastoid operation.

The only way of recognizing the antrum with certainty is in identifying the aditus, and to do this with safety one must see it. There is always danger of dislocating the incus when putting a bent probe or Stacke guide in the aditus, but in the rare cases in which the antrum is difficult to find it is better to risk this and be sure of one's landmarks than to risk damage to the facial nerve. It is the lesser by far of two evils.

Acknowledgement.

I am indebted to Dr. Bryan Foster for permission to publish this case.

Bibliography.

J. E. Frazer: "The Anatomy of the Human Skeleton".

F. W. Watkyn-Thomas: "Principles and Practice of Otology", pages 20 and 347.

Charles Ballance: "Surgery of the Temporal Bone", 1919.

A. Logan Turner and W. G. Porter: "The Structural Type of the Mastoid Process, Based on the Skiagraphic Examination of One Thousand Cases", *Journal of Laryngology and Otology*, Volume —, 1922, pages 27, 115 and 141.

A. H. Cheatle: "Some Points in the Anatomy of the Temporal Bone", Hunterian Lecture, London, 1907, and "Album", 1921.

A. Knick and W. Witte: *"Röntgenologische Studien über die Entwicklung der Warzenfortsatzzellen nach Otitis media im ersten Lebensjahre", Archiv für Ohren-Nasen-und-Kehlkopfheilkunde*, Volume CXIX, September, 1928, page 128.

J. Mouret and G. Portmann: *Acta oto-laryngologica*, Supplement VII, 1928, Copenhagen Congress.

K. Wittmaack: *"Über die Normale die Pathologische Pneumatisation des Schlafenbeins"*, 1918.

Reviews.

A BRITISH MEDICAL ASSOCIATION COOKERY BOOK.

A BOOKLET entitled "Family Meals and Catering"[1] has been issued under the auspices of the British Medical Association. It contains daily menus for three weeks, based on the principles of nutrition enunciated in 1933 by the Nutrition Committee of the Association. This committee had issued a report in which were set out lists of foodstuffs and the quantities necessary for the maintenance of health; the document has now been translated into a form in which it can be understood and used by housewives. This step was taken by the teachers of domestic science at the Summer School of the Board of Education held in London in 1934, who undertook to put into practice the dietary rules formulated by the Nutrition Committee. It must be stressed that the menus included in the booklet are especially designed for the adequate feeding of families on small incomes, and that economy is therefore strictly studied. At the same time it may be mentioned that malnutrition is by no means confined to the poor and that many well-to-do families may be ill-nourished through ignorance of dietary essentials.

The quantities of foodstuffs specified are said to be adequate for a family comprising a man, a woman and three children; the housewife would, of course, use her discretion in adapting these quantities for larger or smaller families. At the beginning of each weekly section the daily menus are stated for two meals—dinner and high tea or supper. On the opposite page are coloured pictures of the prepared dishes, which should certainly appear attractive to the hungry worker. In the following pages the recipes and instructions for the cooking of each item are stated so concisely and clearly that even the housewife with no flair for cookery should be able to create eatable meals. Breakfast dishes are not mentioned, since it is stated that sufficient food should remain from other meals to provide for these. It would be the province of the cook with imagination to make these rehashes bearable and even attractive.

This cookery book, designed for people in the northern hemisphere, may, we think, be equally useful in Australia. Perhaps salads and cold foods might be more extensively used in our long summer months; but it is surprising that the Australian, in spite of a vastly different climate, still for the most part adheres faithfully to the diet of the Mother Country. It may be noted that margarine, so largely used in these diets, is a rare substitute for butter in Australia; but this need not detract from the value of the menus given.

Though this manual serves the science rather than the art of cookery, it is a useful guide for the average housewife, and may be especially recommended to families whose purse is lean and who have to consider ways and means.

[1] "Family Meals and Catering: A Cookery Booklet for Housewives"; 1936. London: The British Medical Association; Australia: Angus and Robertson, Limited. Demy 4to, pp. 27, with illustrations. Price: 9d.

AN INTRODUCTION TO SURGERY.

THE appearance of a third edition of Morison and Saint's book[1] must prove its value, although we feel that the authors have hardly kept to their intention of aiding the student to think out for himself the problems presented to him in the wards and in his text-books. In the earlier chapters this plan is followed, some inevitable difficulties being barely overcome, but in the later chapters a tendency to revert to the standard type of book is found. The introduction of details may confuse the student and can hardly be claimed to illustrate general principles. For instance, the tabulated plan for the treatment of syphilis and the description of the rarely performed operation for psoas abscess are a little out of place, while some of the authors' ideas on the causation of malignant disease will not meet with universal approval. Such inconsistencies are inconsistencies of plan, however, and readers will find in the book a great deal of useful material. Surgical tutors in particular will find much to interest them and to assist them in crystallizing their ideas for presentation to students. A special reference must be made to the numerous illustrations and diagrams, which are excellent.

BOTANY.

MR. BRIMBLE'S text-book, "Intermediate Botany" has been prepared primarily with the object of covering the syllabuses set for intermediate science and certain first year university examinations in botany; but in this admirable and refreshing book the author has achieved a much more important result inasmuch as he has demonstrated that botany is no longer a science of purely academic interest, but is a branch of knowledge of vital importance to human welfare.[2]

The author has presented his subject matter in an interesting and fluent style. He has included in his book an account of such important substances as vitamins and hormones (usually scantily treated) and has given the student as well as the layman some idea of the economic importance of plant life in disease and medicine, aspects which touch human life, welfare and health intimately.

Not only has the author presented the drier and generally duller features of botany in a very refreshing manner, but he has produced a text-book which will be of very great value to student and layman alike. The

[1] "An Introduction to Surgery", by R. Morison, M.D., F.R.C.S., M.A., D.C.L., LL.D., and C. F. M. Saint, M.D., M.S., F.R.C.S., F.R.A.C.S.; Third Edition; 1935. Bristol: John Wright and Sons, Limited. Demy 8vo, pp. 377, with illustrations. Price: 15s. net.

[2] "Intermediate Botany", by L. J. F. Brimble, B.Sc.; 1936. London: Macmillan and Company, Limited. Crown 8vo, pp. 570, with illustrations. Price: 8s. 6d. net.

experimental and practical aspects are strongly emphasized, and such subjects as "Enzyme Action and Digestion", "Plant Products", "The Manufacture of Plant Food" are very well treated for an elementary text-book.

One of the most pleasing features of the book is the frequent reference to the great names which have played so important a part in the development of the various branches of botany. The work is extremely well written in a clear and readable style; it emphasizes the practical and economic importance of botanical science and must occupy an important place amongst the modern text-books on botany. It is profusely illustrated with drawings from the author's pencil, as well as by appropriate photographs, which help the reader to understand the subject matter.

The author is to be congratulated on his successful attempt to produce a work which will prove of great value to all who are interested in plant life.

ABORTION.

"ABORTION, SPONTANEOUS AND INDUCED", by Frederick J. Taussig, is a book which covers a much wider field than the title would lead the reader to expect.[1] It is the outcome of much personal investigation and of a wide knowledge of the literature of the subject. Dr. Taussig has long been interested in the study of abortion, birth control, midwifery and diseases of women, and is exceptionally well qualified for the task of writing a book on this very important subject. When we consider that the number of abortions from all causes in many civilized countries is nearly as great as the number of full-time confinements, when we remember the extent of the morbidity and the not inconsiderable mortality following abortions, however caused, and when we remember that at present the teaching of students in this important branch of work is often inadequate, we realize that this book supplies a long-felt want.

Dr. Taussig has collected and collated a vast mass of literature which has accumulated on the many subjects relative to his task, as the lengthy bibliography at the end of the volume indicates. The only Australian authority quoted is Worrall, who stressed the importance of this subject in an article in THE MEDICAL JOURNAL OF AUSTRALIA some years ago. The book deals with every conceivable aspect of abortion, including medical, social and medico-legal. There is also one chapter, written by Professor W. L. Williams, of Cornell University, devoted to abortion in animals.

The book is divided into four parts: (i) "History and Background", (ii) "Spontaneous Abortion", (iii) "Induced Abortion", (iv) "Social Aspects of Abortion". Part II deals with the anatomy of the early stages of pregnancy, the pathology, symptoms, diagnosis and treatment of abortion, and its complications. In this chapter also is included some consideration of induced abortion. Part III deals mainly with medical aspects of therapeutic abortion, also with preventive measures, such as contraception and sterilization. The Russian experiment in legislation has been given separate consideration. There are throughout the book numerous references to other workers on the subject, and detailed statistics of the results of many investigators are given.

There is an appendix consisting of over twenty pages devoted to the statutes relating to abortion in the different States and territories of the United States of America, and also a chaper on the legal aspects of induced abortion, so that the book should find a useful place on the shelves of members of the legal as well as of the medical profession. In dealing with the vexed question of conservative *versus* active treatment in septic abortion, the author advocates a few days' delay as being likely to increase the resistance of the patient and to diminish the virulence of the causal organism; in no circumstances does he advocate active treatment when there is evidence of spread of infection beyond the uterus. He surveys the mortality rate in nearly all countries, with the exception of Australia, for which apparently no suitable figures were available; he considers that only the tabulation of approximately 1,000 cases justifies any definite conclusions when estimating the mortality rate.

He regards a differential blood count, especially the enumeration of the juvenile and degenerative forms of neutrophile leucocytes, as of great value in estimating the prognosis of septic cases.

For the diagnosis of pregnancy by the hormone test he favours the more speedy Friedman method, in which mature rabbits are used instead of mice, as in the Aschheim-Zondek test. In attempting to cover the whole subject the author often takes up too much space in describing what some would regard as bizarre methods, such as the use of skiagrams of the pelvis after induction of pneumoperitoneum for the diagnosis of early pregnancy. One whole page is devoted to a description of the use of charcoal pencils in the treatment of septic abortion.

Taussig considers puncture of the abdomen to be of considerable diagnostic value in doubtful peritonitis. He is a firm believer in the efficacy of liquid preparations of ergot, which he advocates as a means of determining whether an abortion is complete or incomplete, bleeding and colicky pains being an indication of the latter.

In the section on habitual abortion the author refers to several aspects of this question which seem to have escaped the notice of most authorities, such as the influence of focal sepsis as a causal factor in habitual abortion. Syphilis he regards as being of no great importance as a cause of abortion early in pregnancy, but as a frequent cause of late fœtal death. He refers to two cases of repeated abortion in which a non-venereal prostatitis in the husband seemed to be the only ascertainable cause.

The book is very well printed on good paper and the illustrations are numerous and for the most part very good. One rather serious error was noted on page 217, where the statement is made that there are 250,000 platelets in each cubic centimetre of blood, and on page 207 the word "will" is used instead of "with".

Dr. Taussig is to be congratulated in producing a book which covers the whole field of abortion in the space of about 500 pages. Every obstetrician and every general practitioner, as well as others interested in birth control, will be well rewarded by a perusal of this book.

[1] "Abortion, Spontaneous and Induced: Medical and Social Aspects", by Frederick J. Taussig, M.D., F.A.C.S., with foreword by Robert L. Dickinson; 1936. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay (Surgical) Proprietary Limited. Super royal 8vo, pp. 536, with illustrations. Price: £2 5s.

Notes on Books, Current Journals and New Appliances.

A HOUSEHOLD MANUAL.

IN a world where infallible aids to health and beauty are extolled in the persuasive advertisements of manufacturers, books for the layman on hygiene and first aid need to be compiled with discrimination and care. Sister Parry has written a book of this type[1] in which she displays the common sense of sound experience. Medical practitioners would be happy to see adopted the advice which is given on nursing in the home by untrained people and on the preparation of food for the sick. Of value, too, are the rules for personal hygiene and for the treatment of such emergencies as burns and poisoning. The divers uses to which the everyday inhabitants of the kitchen shelf can be turned should delight the housewife who is not already versed in this lore of old wives' tales. "Do not keep milk in the bathroom or lavatory" is advice which strikes quaintly on civilized ears, but which cannot be gainsaid. This book will find a place in the country rather than in the city home.

[1] "It's in Your Kitchen: Simple Remedies and Hints for Everyone", by A. B. Parry; 1936. Australia: Angus and Robertson. Crown 8vo, pp. 167. Price: 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, AUGUST 29, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE END RESULTS OF THE REMOVAL OF CEREBRAL TUMOURS.

THE removal of an intracranial tumour is one of the most difficult and dangerous of surgical procedures, and it is obvious that, although the tumour may be removed in its entirety and the patient recover from his hazardous adventure, recurrence may take place, or life, as a result of cerebral damage, may be but labour and sorrow or even a useless burden. That the immediate results of the removal of brain tumours are much better than they were twenty-five years ago is a statement that admits of no argument. The change may be partly explained by the discovery and perfection of methods of localization. In the early years of cerebral surgery the surgeon had to rely on the interpretation of signs and symptoms, and these were often misleading. It was no uncommon event, for example, for an exploration to be made over the cerebellar region or over the wrong cerebral lobe when the tumour that was sought was in the frontal region. Most of the credit for the present-day success in cerebral surgery, however, must be given to the enormous advance in surgical technique, and for this Harvey Cushing, America's master surgeon, is largely responsible. The technique of the removal of brain tumours has been discussed in these pages on a previous occasion, but no reference has been made to the end results, in other words, to the justification for the performance of such intricate, time-consuming and extensive operations, for the good reason that little or no information was available on which such a discussion could be based.

From September, 1926, to September, 1927, Hugh Cairns, of the London Hospital, acted as resident surgeon in Cushing's clinic at the Peter Bent Brigham Hospital, Boston. During this period 157 patients suffering from verified intracranial tumours were treated by surgical operation; 22 of the patients died in hospital. The ultimate fate of the 135 survivors has been investigated by Cairns and is the subject of a report that appears in *The Lancet* of May 30 and June 6, 1936. The immediate results of operation on these patients were the subject of a report to the Medical Research Council of Great Britain in 1929. The full report of the present investigation is published in the *Yale Journal of Biology and Medicine* of May, 1936. After the 135 patients left hospital every one of them was kept in touch with the Brain Tumour Registry at New Haven by its registrar, Dr. Louise Eisenhardt; and incidentally it should be mentioned that there are at the registry 2,000 brain tumours, complete with microscopic preparations, clinical histories and follow-up records. Of the 135 patients, 72 have died; 63 are still living from seven to nine years after operation. What Cairns has tried to discover is the period of useful life that the patient has enjoyed after operation; in this way alone, of course, can the real value of surgical treatment of this kind be determined. Cairns points out that to assess the value of surgical treatment it is necessary to consider each type of tumour separately. Not only does the life history of different types of intracranial tumour vary, but in some instances the criteria by which results can be judged also differ. Some patients, for example, may be operated on to conserve sight rather than to save life. The whole group of 135 patients who left hospital alive included 48 who had been operated on for glioma;

eight of these are still alive seven to nine years after operation; five are living useful lives. (The immediate post-operative mortality was greatest among the patients with gliomata; 11 of the 22 deaths that occurred while the patients were in hospital were those of patients with this condition.) Twenty-eight patients of the 135 were operated on for pituitary adenomata; 19 are alive seven to nine years after operation and ten are living useful lives. The meningiomata group includes 26 patients; 18 are alive at the same period of time after operation and fourteen are living useful lives. Of nine patients with acoustic neurinomata, eight are alive and two are living useful lives. In this short account we may group together the craniopharyngiomata, cholesteatomata, blood vascular tumours, granulomata and miscellaneous tumours; 17 patients are in these groups; ten are alive seven to nine years after operation; six are living useful lives. Seven of the 135 patients had been operated on for metastatic tumours, and none of them were living seven years after operation. Thus 63 of the 135 patients were alive seven to nine years after operation and 37 of them were living useful lives. These figures give the barest outline of the end results in the 135 cases. Obviously many of the patients who died in the seven to nine year interval would have received some benefit from operation; and no doubt some of them enjoyed a certain period of relief from all suffering, and possibly of usefulness. For these points the reader is referred to Cairns's paper.

From whatever point of view these results are considered—the purely economic, the social or the scientific—they must be regarded with the greatest satisfaction. The report does not include the best results that the registry at New Haven has to offer. The longest period of survival in the 2,000 cases recorded is more than twenty-six years, and this in the case of a glioma. In other groups the longest periods are twenty-five, twenty-four, twenty-two years, and so on. In the future no doubt the number of long-period survivals will become larger. Two further aspects should be emphasized. In the first place the organization of Cushing's clinic, with its registry and complete follow-up system, is a model that should be followed wherever surgery in any of its branches is practised. Australia particularly has need of an organization of this kind in more than one of its centres. In the second place Cairns's report shows the true spirit of cooperation and research in the pursuit of an ideal. Cairns is well known as one of the chief exponents of neurosurgery in Great Britain, and yet he can find time to visit the clinic of his former teacher to undertake the investigation that we have described. Cairns pays a warm tribute to Cushing and to the way in which he was allowed to examine freely and critically the results of his year's service at Boston. He concludes by writing: "That such unrestricted studies of one man's work by his pupils are possible indicates to some extent the sense of partnership with which Dr. Cushing has invested his clinic, a quality that is the mark of all great teachers."

Current Comment.

BLOOD TRANSFUSION IN TYPHOID FEVER.

TYPHOID fever is one of the infectious diseases that is becoming more or less of a rarity in the larger centres of this country, and the occasional cases seen in the more sparsely settled areas are few in number, so that it does not assume epidemic proportions. Yet epidemics do occur, though fortunately on a small scale, and during the past year or two the cases observed in many parts of Australia have been greater in number than is usual. In any case the typhoid group of infections often causes great anxiety; the disease is prolonged and often severe, its complications are sudden and dangerous, and its treatment is still in the main a question of the apt handling of the patient by skilled nurses. The more liberal feeding of the patient and the more thorough hydrotherapy (as experiences in Brisbane have shown us) have improved the outlook, but probably no other safe and lasting additions to therapy have been made. Pedro T. Lantin and Fortunato S. Guerrero now present a promising series of cases from Manila in which they have employed blood transfusions.[1]

In a previous communication these authors reported good results from transfusion in a series of ten patients severely ill with typhoid fever, and to this number they have added forty-one more. Controls were provided by treating a group of thirty-four patients without transfusion, and both groups were selected from the same wards at the same time, care being taken that the patients in

[1] *The American Journal of the Medical Sciences*, June, 1936.

each group were suffering, so far as could be judged, from illness of equal severity. The method used was to withdraw 150 cubic centimetres of blood from the patient and immediately to replace this amount by transfusing 250 cubic centimetres of blood from a healthy donor. If no favourable reaction was observed or if the toxic state of the patient returned, the blood exchange was repeated on the following day and on succeeding days if necessary. As many as seven transfusions have been given to one patient. In cases in which intestinal hæmorrhage occurred, 200 cubic centimetres of blood were transfused without any previous bleeding of the patient.

The rationale of this treatment is based by the authors on the assumption that the blood of the typhoid patient contains toxins the removal of which may improve his clinical condition, and also that the introduction of fresh blood will bring an added supply of protective elements. Further they felt that the risk of hæmorrhage might be less owing to an anti-hæmorrhagic effect inherent in transfusions. Whether these arguments are sound or not is not clear in the present state of our knowledge. But the authors certainly present encouraging figures, for their mortality rate in the treated cases was 24%, whereas it was 47% in the untreated. It will be observed that they were apparently dealing with a very toxic type of infection, though it is well known that the death rate in typhoid is very variable, not only from epidemic to epidemic, but even from month to month during the same outbreak. Attention is drawn to this communication, as the method might be worthy of trial.

BISMUTHIA.

IT is well known that the prolonged administration of silver salts can cause permanent pigmentation of the skin and mucous membranes, and the name "argyria" is at least familiar even if the condition itself is very rare. Silver salts, once popular to some degree as an internal remedy, are now practically never prescribed, except for external use, so silver pigmentation is not likely to be seen in the future. But now a similar condition has been described following the prolonged administration of bismuth salts in large quantities, and for this the name "bismuthia" has been coined. H. C. Lueth, D. C. Sutton, C. J. McMullen and C. W. Muehlberger have encountered a man suffering from this form of pigmentation, which does not appear to be mentioned in the literature available.[1] This patient entered hospital complaining of severe diarrhœa, which had troubled him intermittently for eighteen years, and his remarkable complexion at once attracted notice. The skin was coloured deep bluish grey with a distinct metallic sheen, and the mucous membrane of the mouth and pharynx was described as being a deep indigo-purple-black. The hair was deep mahogany colour, and these remarkable changes gave the patient a strangely cadaveric appearance, enhanced by the grey conjunctivæ. The pigmentation was universal, but was most intense over the forehead, the face and neck, and dorsa of the hands; the unexposed skin surfaces were of a drab ashen hue. The patient was under the impression that he had been given silver nitrate for a supposed gastric ulcer eighteen years previously, but subsequent investigation proved beyond doubt that the metal deposited in the skin was bismuth and not silver. Before adverting to the investigations carried out, it is of interest to note that this man had suffered from a chronic colitis for eighteen years, which began shortly after an accident in which he sustained a head injury of considerable severity. This conjunction has been recorded in the literature before and recalls the work of Cushing and others on the relation of lesions of the interbrain to the production of acute ulcer of the upper alimentary tract.

The patient gave the history that after two years of medication with various salts of bismuth he noticed a steely-grey colouration of the skin and stated that a few days after this his skin assumed the permanent colour described by the authors. The doses of bismuth salts taken ranged from 10 to 30 grains, but the total amount ingested in a day was sometimes as high as 600 grains. Further examination of the patient revealed ulceration of the duodenum, but no other obvious change in the digestive tract; later a tuberculous lesion was discovered in the lungs. Injection of a solution of sodium thiosulphate and potassium ferricyanide into the skin over a test area caused blanching; this is also observed in argyria. Biopsy of a piece of skin showed innumerable metallic granules in the dermis, and chemical tests demonstrated the complete absence of silver, lead and copper, and proved the metal to be bismuth. No bismuth could be found in the urine.

This extraordinary case raises the question of the entire harmlessness of bismuth. Symptoms of bismuth intoxication are not very rare, and even death has occurred. The authors of this article point out in their review of the relevant literature that bismuth subnitrate, when taken by mouth, can cause toxic symptoms by liberation of nitrites. Further, dusting powders and pastes have caused poisoning, and dermatoses and gingivitis have been frequently observed after the parenteral administration of various preparations of bismuth. But in the present instance only the insoluble salts were used, and they were taken by mouth; it is suggested that chronic ulceration of the colon had perhaps altered the rate of absorption. Though surely this condition must be excessively rare, it shows that it would be wise not to encourage patients to take unlimited quantities of even an insoluble bismuth salt over long periods. Since very large doses of bismuth subnitrate have been recommended for the tropical dysenteries and ulcerative colitis, perhaps caution will not be misplaced.

[1] *Archives of Internal Medicine*, June, 1936.

Abstracts from Current Medical Literature.

RADIOLOGY.

Pulmonary Mycotic Infections.

RAY A. CARTER (*Radiology*, May, 1936) states that pulmonary mycotic manifestations on the X ray film vary from case to case and will simulate closely some manifestation of tuberculosis. On the whole, minority characteristics of tuberculosis are simulated. These are seen so much more frequently as tuberculosis that they are properly recognized as tuberculosis-like. However, they are definite mycotic predilections. With persistent failure to find the tubercle bacillus, and with suspicious peculiarities of history, the chance of mycosis is sharply increased. Geographic location, occupation, associated extrapulmonary lesions or certain characteristics of the film of the chest may direct suspicion to mycosis or to a particular disease, such as blastomycosis, coccidiosis or actinomycosis. In coccidoidal granuloma the primary infection is cutaneous, pulmonary or, rarely, oro-pharyngeal. The digestive tract is practically immune. Pulmonary onsets are very common; their full proportion is not known, because they may subside and become significant only by later metastatic manifestations. The disease is essentially granulomatous, its basic lesion a tubercle resembling that of tuberculosis. Lymph glands are selectively involved. Abscess is common. The lesions are quite invasive, but blood vascular metastatic dissemination is more prominent than spread by contiguity. Intrathoracic lesions include caseous enlargements of tracheobronchial and mediastinal glands; infiltrations, diffuse miliary, nodular and nodal; consolidations, small to massive; abscesses; pleural exudates, local or general; and associated lesions of thoracic, osseous and soft structures. A few air-containing cavities may occur and are mostly of the small acute type, not over two centimetres in diameter, and are like recent excavations of small tuberculous caseations. Their small numbers and, more often, their absence from films are striking. No large fibrous-walled cavities are seen. This sparsity of cavities is noted in spite of numerous necroses reported at autopsy. Apparently bronchial drainage is not readily established. Mediastinal adenopathy and hilar enlargement are much greater and more frequent than in tuberculosis. Descending bronchiogenic spread from an older apical or subapical lesion does not occur in the mycoses as in tuberculosis. The common tuberculous manifestation of retraction of the mediastinum towards the lesion does not occur in the mycoses. Miliary manifestations are much more indefinite on the films than in tuberculosis. Any of the mycotic appearances that the author has seen could have been due to tuberculosis. X ray findings heightening the chance of the mycosis are: mediastinal and hilar adenopathy in the adult; vague miliary infiltration; absence of descending spread, of cavities in advanced lesions, and of elder subapical lesions; destructions of bone or abscess in the thoracic wall, or entirely extrathoracic lesions. Manifestations, absent or infrequent in the mycosis and decreasing the chance of its presence, include the following: linear fibroses, except as they may be present incidentally; mediastinal or hilar retractions; air-containing cavities, particularly fibrous-walled, large round thin-walled or numerous small acute cavities; parenchymal calcifications; obvious descending spread; acinous nodose manifestations. In blastomycosis the most frequent involvement is a characteristic granulomatous lesion, spreading by contiguity in the skin, with only rare systemic metastases. The occasional systemic infections are strikingly like those of coccidoidal granuloma. In actinomycosis the primary infection may be pulmonary, cutaneous, oral, œsophageal or intestinal. Involvement of the digestive tract is common, in contrast to blastomycosis and coccidiosis. Pathologically the disease is a granuloma, having a tendency to fibrosis and abscess. Metastatic spread is less frequent and less extensive than in coccidiosis and blastomycosis. Direct invasive extension is more prominent. Thus the disease tends to produce one or a few extensive lesions rather than numerous scattered small foci, as in the other mycoses. Abscesses, cutaneous, subcutaneous or deep, are prone to burrow extensively. Lesions of the bone and joint occur, which may be metastatic, but which are frequently extensions from adjacent soft tissue involvement. Pulmonary lesions have been classified as broncho-actinomycotic, pleuropulmonary and pneumo-pulmonary. The first may be considered an actinomycotic bronchitis, somewhat akin to the mild pulmonary mycoses, bronchomoniliasis and aspergillosis. In this rare form the usual invasive spread through pulmonary structures does not occur. The pleuro-pulmonary type is featured by fibrosis, abscess and granulomatous consolidation involving both pleuræ and adjacent lung, so that they are "glued" together by dense fibrotic tissue to such a degree that they are merged into one inseparable mass. Such lesions may be massive or local. The pneumo-pulmonary type, starting in the lung itself, develops to consolidations, usually massive, combined with abscess, miliary or nodular infiltration and fibrosis. The pleura may or may not become involved. Discrete nodular involvement may occur without consolidation. Röntgenographically there is a preponderance of actual consolidations, local or massive. Abscesses may or may not be demonstrable. Pleural thickening is often extreme. Empyema occurs as local encapsulations or general effusion. A clear record of extension to or from the thoracic wall is often obtained. Discrete nodular involvement, from miliary to coarse, may be present, simulating miliary tuberculosis, or, as cited by Pancoast and Pendergrass, nodular pneumonokoniosis. Widespread interstitial fibrosis without consolidation may occur. Suspicions of actinomycosis as against tuberculosis are a predominance of plastic pleural or empyemic manifestations, directly spreading invasiveness of consolidations, extensive lesions confined to one side of the thorax, and particularly a clear record of extension through the thoracic wall, either from within or without. Streptotrichosis may be considered to be involvement by the allied organisms, sporothrix, streptothrix and pseudoactinomyces. Skin, mucous membranes or lung may be primarily affected. Röntgenographically, the disease closely resembles actinomycosis. In torulosis the portal of entry is more obscure because of its usually chronic character. Primary respiratory onset is frequent. The disease may be local or general; the former is particularly chronic. In the latter, eventual fatal meningeal involvement is usual. Pulmonary involvement is usually chronic. Consolidations, essentially nodular in structure, are frequent, and there is a strong tendency to fibrosis, but little to caseation. The monilias are occasionally found in tuberculosis-like pulmonary disease, clinically and radiographically, or in symptomatic asthma, with no other organisms found in the sputum to explain the lesions. While moniliasis is considered a mild mycosis, the disease varies from mild chronic bronchitis to severe or fatal tuberculosis-like illness.

Radiology in Obstetrics.

R. E. ROBERTS (*British Journal of Radiology*, July, 1936) states that, provided the radiographs are of good quality, a fœtus of anything more than sixteen weeks' gestation should in all cases demonstrate its presence on the films. In favourable cases an earlier gestation than this may be shown. In intrauterine death there usually takes place, within a period of a week or less, a shrinkage of the brain substance, with a consequent falling in of the cranial vault. As a result, in such cases the radiographs may reveal an overlapping of the cranial bones. This appearance is reputed to be present often as early as four days after the death of the fœtus, but precision on this point is impossible, owing to the difficulty of fixing the exact date at which death takes place. In a case in which fœtal death is suspected and in which this sign is not present, further X ray examinations should be made at weekly intervals for two to three weeks. If in the resultant radiographs there is no evidence of over-

riding of the cranial bones and there is evidence of fœtal growth, the fœtus is obviously alive. If, on the other hand, either the sign becomes positive or there is seen to be an absence of growth (or even a shrinkage from falling in of the thorax or increased flexion of the spine) or immobility of the fœtus, the death of the fœtus is indicated.

Emphysematous Blebs and Bullæ.

EUGENE FREEDMAN (*American Journal of Roentgenology*, March, 1936) states that although both blebs and bullæ represent localized air pockets within the lungs, they differ from each other as far as location is concerned. Blebs are situated immediately underneath the pleura, while bullæ are in lung tissue itself. In a healthy person spontaneous pneumothorax is caused by the rupture of an emphysematous bleb or bulla and is not due to latent non-demonstrable tuberculosis. Emphysematous blebs and bullæ are often recognizable on the skiagram as localized areas of increased brilliancy in the lung fields, surrounded most commonly by hair-line-like curved and annular shadows, which are due to a zone of atelectasis in the neighbouring alveoli caused by the presence of the air pockets. The lungs need not show the radiological evidence of generalized emphysema, since blebs and bullæ are occasionally found in the absence of a generalized emphysema. In the latter case the air pockets are usually secondary to localized scar tissue. Attacks of asthma or of strenuous cough, aortic aneurysms, mediastinal or lung tumours, patches of atelectasis or of pneumonic consolidation may lead to the formation of blebs and bullæ by interfering with the normal respiratory mechanism. Occasionally an emphysematous bleb or bulla may closely simulate a tuberculous cavity.

PHYSICAL THERAPY.

X Ray Therapy of Thromboangiitis Obliterans.

G. F. PFAHLER (*American Journal of Roentgenology and Radium Therapy*, December, 1935) discusses the ætiology, history and symptoms of *thromboangiitis obliterans* and then relates the effect of X radiation to the sympathetic nerve ganglia indicated by the location of the disease. He uses a kilovoltage of 200, and treatment is given three times a week till a full erythema dose is obtained. Pain is relieved usually in about two to three weeks after the inception of the treatment, and at times relief occurs more promptly. Of the fifty patients reported, only 10% failed to obtain relief. If nothing else could be accomplished, the author considers this a great triumph. One of the patients could walk only a city block when he would have to stop on account of pains in the calf of the legs. He has been completely relieved and three years later could play 36 holes at golf at the age of sixty-seven. Some of the patients treated have had to give up their work as long as three years before treatment and have returned to their work after treatment. The relief of special symptoms is stated to have been as follows: Intermittent claudication disappears sufficiently within two weeks for the patient to walk without distress, and in 50% of the cases reported these symptoms disappeared completely within six weeks. Circulatory and trophic disturbances improve in from four to six weeks. The normal colour reappears and the cyanosis disappears. The limbs become warmer and are better able to withstand changes in temperature. Phlebitis shows improvement early. Active signs of inflammation disappear with supervening signs of resolution. Venous and lymphatic stasis, if present, disappear rapidly. The pulse, when absent at the beginning of treatment, is not recorded as reappearing in any of the cases. Therefore the author infers that the occluded artery is not restored to normal, but that the improvement must be due to an establishment of collateral circulation. Ulcerations show a tendency to improve within a few weeks and disappear within a few months. Granulations appear and epithelialization of the margins is noticed early, and an appearance of an ordinary ulcer is soon established. Marked general improvement appears rapidly, chiefly owing to the relief of pain, and therefore the patient obtains increased rest and sleep. The cachectic colour and the agonized expression disappear. The patients gain in weight, look rested and become hopeful.

X Ray and Radium Treatment of Tumours of the Conjunctiva.

GUSTAV PETER (*Radiology*, December, 1935) states that in a previous paper he brought forward results which showed that the β rays of radium, which are the least used in present-day practice, are the most active. In one case, however, that of a boy of fourteen, a glaucoma developed with the use of radium. This has caused the writer to undertake the use of X radiation in this condition, and he has found that with a comparatively low kilovoltage (44 kilovolts) excellent results have been obtained without any damage to the eye itself. Exact physical measurement is given in the text of the dosage used, and a table showing the technique in each case is reproduced.

X Ray Treatment of Gas Gangrene.

JAMES F. KELLY (*Radiology*, January, 1936) records a series of thirty-four cases of gas gangrene. Thirteen patients were operated upon; five of them died. Of the remaining twenty-one with extremity involvement, who were not operated upon, none died. In the group not operated on treatment consisted in the use of serum, local antiseptics and small doses of X rays given frequently. The author considers that treatment should be started regardless of how hopeless the case may seem. Treatments should be given morning and evening, or twice a day. Sufficient voltage should be used to insure penetration of the involved tissue; about 90 to 100 kilovolts should be used for the extremities, from 130 to 160 kilovolts for the trunk, and about 100 *r* units per dose. Amputations should be discouraged, except in those instances in which the tissues are hopelessly damaged by causes other than gas gangrene. The dark appearance does not indicate a true gangrene and clears up after successful radiation.

Uterine Myomata.

LUDWIG FRAENKEL (*British Journal of Radiology*, May, 1936) discusses the treatment of uterine fibromata. Although he is a surgeon and his surgical experience of the operative treatment of uterine fibromata has been satisfactory, he admits, in fact he insists, that in the large majority of cases the irradiation treatment of these conditions is not only equally satisfactory, but is actually the best method of treatment. This means that the bleeding is stopped, that the tumour is reduced to such a size that it does not cause inconvenience, and that the patient is quite well. The malignant degeneration of a myoma either before or after irradiation is unknown in his experience. He does not believe that such degeneration is possible, but that all cases that have been so diagnosed are in reality unrecognized primary sarcomata. He does not know of any special group of myomata that cannot be cured by irradiation. There are no operative risks, particularly of that terrible "bolt from the blue", embolism. There is no post-operative pain, *Wundschmerz*, and there is much less cost to the patient. In the hands of an experienced radiologist there are now no radiation risks. Scientific accuracy of dosage has replaced the empirical and inaccurate use of radiation, which was common until recent years. The technique must be in the hands of a competent radiologist who will work in conjunction with the gynæcologist. In spite of the success of irradiation, however, there are still a large number of cases in which operation is necessary. Irradiation treatment achieves its results indirectly by putting the ovaries out of action. For many reasons it may be undesirable to do this, and operation may be the method of choice for various reasons. The patient may desire to marry and have children. Irradiation treatment may interfere with sexual desire and intercourse. If there is no history of insanity in the family, irradiation treatment should not be carried out, for it amounts to castration; then operation becomes the lesser risk, because some part of the ovarian tissue can be left.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on April 30, 1936, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. E. H. M. STEPHEN, the President, in the chair.

Radiology and Heart Disease.

DR. KEMPSON MADDOX read a paper entitled "Radiology and Heart Disease: The Medical Aspect" (see page 290).

DR. K. B. VOSS read a paper entitled "Radiology in Relation to Heart Disease" (see page 296).

PROFESSOR C. G. LAMBIE expressed his appreciation of the papers and said that radiology had advanced cardiology and provided a valuable means of making the diagnosis more precise. If it was resorted to more often, conditions at present overlooked would be recognized. It was unfortunate that radiology in this field was not so extensively used as it was in Continental countries. One of the reasons why radiology was not more frequently resorted to was the lack of facilities. Physicians, especially in hospitals, ought to avail themselves of the opportunity of inspecting for themselves the shadows cast by the heart; they would thus gain the experience necessary for the interpretation of these shadows.

Physical signs, from the time of Laennec, had been found to be open to fallacies, and radiological examination had further revealed these fallacies. The use of radiological examination was to be regarded as essentially an extension of the method of inspection.

Professor Lambie said that it had been remarked that the present age had not sufficient faith. Yet, while seeing was believing, it was still possible for the medical practitioner to be led astray, because, after all, in radiological examination he was only seeing shadows and not directly observing anatomical structures. The danger was that faith might see too much. The shadows seen by X rays, like the time-honoured physical signs, required interpretation, and those not familiar with this world of shadows could easily be deceived. Even at this present meeting Professor Lambie had been sceptical of some of the things demonstrated. However, he was of the opinion that radiological examination was a most valuable adjunct to clinical examination.

DR. O. A. DIETHELM congratulated Dr. Maddox and Dr. Voss on their expositions of the subject and expressed his appreciation of the amount of work that the papers represented. Advances in radiology had been of the greatest assistance in clinical work. These advances should provide an impetus to clinicians in the future to gain experience in this field and thus to add to the methods of diagnosis. Such work was of the greatest assistance to physicians, who should take advantage of any facilities that offered in order to become expert in this direction; they would certainly do so in the future. Physicians were in a happy condition, having at their disposal the history of the patient, the clinical information and also the assistance of screening and fluoroscopy. In the future the diagnosis must be made more exact.

DR. H. M. CUTLER congratulated the speakers. He said that he was not able to appreciate Dr. Maddox's paper, but he could appreciate that of Dr. Voss. Dr. Maddox had stressed the question of distortion. By the present method of screening there must always be distortion. But the radiologist got into the habit of allowing for the distortion and the difficulty was thus overcome. The distance for taking films varied from one to two metres. It was pointed out that for a distance of over five feet the relative degree of distortion was so small that five feet was the best distance at which to get detail of the lung fields and heart shadows, when the various technical requirements at greater distances were considered.

Dr. Maddox remarked that if he were to lose his stethoscope, examination by the fluoroscope would be the best method. This was hard to credit. It was necessary to correlate all methods. The whole trend of the present meeting had been to gain all the clinical facts. Was it best to examine hearts radiologically or clinically? Dr. Cutler thought that a combination of both methods would produce the best results.

DR. E. W. FRECKER said that he had enjoyed the addresses. The speakers had attacked the problems that attended diagnosis and treatment from the viewpoint of the radiologist and physician respectively. The radiologist tried to ascertain the facts of internal anatomy by a method of direct visualization. The physician, on the other hand, tried to ascertain the same facts from the clinical signs and patient's history. His deductions were therefore more indirect than those of the radiologist, which were based on the demonstration of real anatomical structure. The radiologist could thus be of great assistance to the physician in the diagnosis of heart conditions.

Any attempt to make a hard and fast diagnosis from films alone was doomed to failure, as the clinical features were paramount and necessary for interpretation. The physician and the radiologist must therefore come together. In some ways it was ideal for the one man to be both radiologist and physician; but it was seldom practicable. To many physicians the handling of apparatus was not congenial, and to most others apparatus was not available. The professed radiologist must therefore always exist to carry out this special help. At present radiologists were not often asked to examine the heart alone. Most of their findings in relation to the heart arose incidentally during the examination for other conditions. Screening of the chest before an examination of the alimentary tract often revealed unsuspected heart abnormality as a cause of digestive disturbance. Dr. Frecker had himself on two occasions, when examining chests of patients diagnosed as suffering from pneumonia, found large and unsuspected pericardial effusions. It was always necessary for the radiologist when he received a commission from a physician to be given full details concerning the patient. Only thus could the examination performed by the radiologist attain full efficiency.

DR. A. S. WALKER said that Dr. Frecker had summed up the position admirably. It was ridiculous to expect the radiologist to diagnose cardiac lesions without a report from the physician. Yet the physician's examination might be inaccurate and might be supplemented by the findings of the radiologist.

It was interesting to see fluoroscopy of the heart performed in London, where such men as Parkinson were both expert clinical cardiologists and expert interpreters of X ray shadows. In Australia no one saw a sufficient bulk of work to become so skilled as this. But Dr. Walker himself found it interesting to screen cardiological cases in hospital. It enabled the coordination of the eye and ear and the correction of clinical impressions, and there was no need for the physician thus gaining experience to pretend to be an expert cardiological radiologist.

Dr. Voss had taken a sound point of view in his comments on the mathematics of the Vaquez school; they were really an exaggeration. What was experimental error? It was easy to assume a pseudo-accuracy, as pointed out by a teacher of mathematics who said that the candidates for an examination, in working out the height of a lighthouse, had in many cases obtained a result allegedly correct to the thickness of a sheet of writing paper. Dr. Voss rightly relied more upon the qualitative than on the quantitative.

Dr. Walker wished that he had with him the films taken of a patient at the Royal Prince Alfred Hospital. On the films there had been a definite lesion clinically and radiologically at both apices, which were undoubtedly tuberculous. The outline of the heart had suggested a pericardial effusion. The patient was only moderately ill and was not in bed. After some months the outline changed so much that it was difficult to believe that the films were both of the same patient; but the lesion in the lung was the same and the film was of the same patient.

Dr. Walker asked if anyone had seen a picture of tuberculous pericarditis that had spontaneously subsided.

DR. D. G. MAITLAND said that, from the radiological standpoint, the fact should be more stressed that radiologists were clinicians and not merely radiographers. Radiologists frequently received a request to make an X ray examination of a heart and were given no clinical details at all. A radiologist was dealing with shadows, and shadows were subject to distortion. Therefore it could not be said that an X ray film was a photograph. Still the shadows were as perfect as radiologists could get them, since standard positions were used. In obese patients the ordinary clinical methods could not give adequate information as to the size, shape and position of the heart, and in such circumstances X ray examination was illuminating and possibly the only method of estimating these facts. Dr. Maitland recalled the case of an obese woman with complete transposition of viscera. She complained of pain on the left side of her chest, and the clinician had asked for an examination of her heart without suspecting dextrocardia. X ray examination in obesity, therefore, was a definite advantage over the purely clinical method. Dr. Maitland thought that screen examination of the heart, assisted by radiography at a distance, would give all the required information without further delicate mathematical methods, which had been proved to be only approximately correct.

Dr. Maddox, in reply, said that in this field the overlapping between cardiology and radiology was very considerable. It was natural that the cardiologist would develop an efficiency in radiology satisfactory for clinical purposes, just as the urologist and chest physician did in their particular spheres. The cardiologist would turn to the radiologist for help in the presence of an extracardiac shadow. The subject of cardiological radioscopy was not difficult; it was a matter of having the opportunity for studying it. Workers in large cardiac clinics, such as Parkinson, saw an immense amount of work and thus soon became skilled observers. Most physicians had to rely on seeing odd patients in X ray departments of public hospitals. In this way it took a long time to become conversant with radiology of the heart. The expense of a radiological unit was considerable. It was for the physician himself to decide whether his interest in cardiology was sufficient to warrant the expense. Dr. Maddox said that a short post-graduate course in this subject would put many clinicians on their feet and give them far more confidence in interpretation. As to the question of the stethoscope *versus* the fluoroscope, Dr. Maddox said that he had no intention of losing his stethoscope. He merely thought so highly of fluoroscopic examination that, if he were provided with the clinical history only, he would prefer this method.

In conclusion, Dr. Maddox thanked those who had taken part in the discussion.

Dr. Voss, in reply, mentioned Professor Lambie's scepticism regarding the films of coarctation of the aorta that had been shown. In both cases the diagnosis had been made clinically and the X ray examination had been made only to confirm the clinical findings; radiologically the films were in accordance with the clinical findings. Dr. Voss did not show the oblique films showing the actual stenosis, because he did not think that the condition would show on the screen. In both cases the clinical findings favoured and did not oppose the radiological appearances.

Dr. Cutler had discussed the question of distortion. Dr. Voss said that at a distance of from six to seven feet the distortion was only about 0·7 millimetre, which was about the thickness of the line drawn at the side of the heart to measure it. This slight distortion was well within the limits of experimental error beyond which accuracy could not go.

Dr. Walker had mentioned films taken at the Royal Prince Alfred Hospital. Dr. Voss said that there was a similar series at the Royal Alexandra Hospital for Children, of a child with pericardial effusion. It had been interesting to watch the heart shadow getting bigger and then smaller. A needle was not inserted. The diagnosis of pericardial effusion was made clinically as well as radiologically; subsequently the child was discharged from hospital.

Dr. Stephen said that he had found the skiagrams fascinating and would like to see them again in a private audience. He thanked Dr. Maddox and Dr. Voss for their attractive papers.

A MEETING of the Queensland Branch of the British Medical Association was held at the Mater Misericordiæ Children's Hospital, South Brisbane, on May 14, 1936. The meeting took the form of a series of demonstrations by members of the honorary staff.

Anterior Poliomyelitis.

DR. A. V. MEEHAN showed four patients suffering from recent attacks of anterior poliomyelitis, and demonstrated the methods of splinting and reeducation that were being used.

It was pointed out that the affected limbs and the body were put at rest in a plaster bed and that the cast did not enclose the body and limbs. Muscle reeducation was carried out by trained masseuses twice a day. The masseuses on the staff of the hospital were seen at work and the details of reeducation were shown.

The reeducation was commenced as soon as the limbs could be handled without pain, in one case on the day after the patient's admission to hospital, in another in five days, and in a third in nine days. Each muscle was put through as much exercise as it would stand without fatigue. The muscles at first were not asked to work against gravity, and in a very weak muscle the friction of the limb in movement was as far as possible eliminated by resting it on a polished board dusted with powder. As power increased, the muscles were exercised against the force of gravity and later against stronger resistance. The splinting prevented deformity and protected the weak muscles against the weight of gravity and the pull of opposing muscles.

"Trick" movements, in which active muscles were encouraged to usurp the function of the muscle being reeducated, by giving rise to a false jerk instead of a smooth, natural movement, were sedulously avoided. It was pointed out, for instance, that a child with a paralysed deltoid and intact trapezius and biceps muscles could be readily taught to jerk the arm upward momentarily in abduction. This movement was a favourite one for unqualified practitioners to teach a child and was rather impressive to onlookers. It was not a useful movement, but merely a "stunt", and it did harm by discouraging the patient from concentrating on the development of true deltoid movement.

Dr. Meehan's first patient was a female child, aged three and a half years, who was admitted to hospital on March 11, 1936, with a history of having become feverish and drowsy five weeks previously. This continued at intervals for one week. The child was in bed only while feverish, never for a whole day. Then, on walking, the child limped; she was splinted for ten days, then was put to bed with the leg between sandbags.

She walked with a rather well-marked right "hip" limp and the right lower limb was internally rotated. There were no deformities. All the muscles of the right lower limb had subnormal power, though they could act against slight resistance. The glutei were the weakest, but they could act against gravity.

On March 12, 1936, a scrim cast was applied to the body and right lower limb with some hip abduction, slight knee flexion and the foot at 90°. Muscle reeducation was commenced twice a day. On May 9, 1936, the child was out of plaster for half a day. Muscle power was increasing. On May 14, 1936, all muscles were acting strongly against resistance. The child was allowed out of plaster cast for half of each day, and walking was to be commenced in four weeks.

Dr. Meehan's second patient was a child, aged four years, who was admitted to hospital with a history of having been well till five days previously. On April 7,

1936, he had been off colour, complaining of nausea and anorexia; he was slightly feverish. Next morning he was worse, complaining of headache and pain in the legs; he was constipated. After that his condition improved a little and then a limp developed in the right leg. He was admitted to hospital on April 12, 1936. On examination it was found that if the child attempted to walk the knees became flexed and the legs were inclined to collapse under the weight of the body. In the right leg all the muscles were acting, but there was some paresis. In the left leg there was paresis of the quadriceps, *tibialis anterior*, *extensor hallucis* and *extensor digitorum*. There was some rigidity of the spine and the abdominal muscles were very weak.

Lumbar puncture yielded a clear fluid which was under a pressure of 200 millimetres of mercury and which contained 20 cells per cubic millimetre. On April 16, 1936, a plaster scrim cast was applied to the body and lower limbs. On April 21, 1936, muscle reeducation was commenced. On May 8, 1936, the left quadriceps had good power; no *tibialis anterior* was detected, the *extensor hallucis* and the *extensor digitorum* had moderately good power, the *tibialis posterior* was weak. The right leg showed all muscles acting, the quadriceps weakly. The *erector spinæ* and abdominal muscles were acting weakly. On May 14, 1936, the child was still in the scrim cast, having reeducation twice a day. All muscles could then act against resistance except the *tibialis anterior* on the left side, which was active but weak.

Dr. Meehan's third patient was a boy, aged three years and nine months. He was admitted to hospital on April 26, 1936, with a history that he had been well till the day previously, when the mother had noticed that he could not walk without swaying the body, and if he were left to stand for any length of time he would fall. He complained of occasional pain in both legs, and he was constipated. There was no rise in temperature. Lumbar puncture revealed slightly turbid fluid under 280 millimetres of water pressure. There were three cells per cubic millimetre; no organisms were present and culture was sterile. On April 26, 1936, the left leg showed weak flexors of the hip and a weak quadriceps. Otherwise all the muscles were acting fairly well, though there was a suggestion of slight weakness in the whole body. The right leg showed some general paresis, with paralysis of the *tibialis anterior*. The spine showed marked rigidity. On April 28, 1936, a plaster cast was made for the body and lower limbs. On May 1, 1936, muscle reeducation was commenced. At this stage the left leg showed that all muscles below the knee were good. The peroneals were acting fairly well. The quadriceps showed definite weakness and barely worked against gravity. The flexors of the hip were weak; the abductors of the hip could not hold the leg against gravity; the *gluteus maximus* was very weak but was just acting. In the right leg, below the knee, there was no power in the *tibialis anterior*. The quadriceps was acting well, but could not completely hold the leg against gravity. The flexors of the hip were acting fairly well; the abductors of the hip did not quite hold the leg against gravity. The *gluteus maximus* was weak. The abdominal muscles were weak but acting; spinal rigidity was still present. On May 14, 1936, it was found that all muscles acted against some resistance, but the right quadriceps tired after prolonged treatment. Reeducation and splinting were being continued.

Dr. Meehan's last patient was a girl, aged six years, who was first seen on April 29, 1936. She gave a history that three months previously she had become feverish, had vomited and had had headache and pain in the neck for about three days; it was then noticed that the left arm could not be raised. The arm had been weak ever since, and the only treatment was home massage. On examination there was wasting and complete paralysis of the left deltoid and spinati. There was just a flicker of power in the left biceps, but no active power of elbow flexion. The internal rotators were working well, but there was no power in the external rotators at the shoulder. A splint for the left arm, of the birth palsy type, was ordered. On May 14, 1936, the arm was on the splint and the child was having muscle reeducation. There was definite power in the deltoid, though not against resistance, and there was good power against slight resistance in the elbow flexors. The external rotators of the shoulder joint were active, but no power was detected in the rhomboids.

Chorea.

DR. P. A. EARNSHAW showed a girl, aged eight years, who had been admitted to hospital a year previously, after coming from Ayr in north Queensland. Unfortunately the history was not satisfactory, as the mother was a very unobservant person. The child was quite well till April, 1934, when she developed purposeless movements and would stumble and fall. Her teacher noticed this for three months before the mother was aware of it. She was in Ayr Hospital for two months, and later was treated in the out-patient department of the Mater Misericordiæ Hospital for three months. She was admitted as an in-patient on May 10, 1935. She walked into hospital, was very clumsy and would stumble when excited. The mother had suffered from chorea when the same age as this child, and it continued until the menses commenced at twelve years of age.

On admission the patient had choreiform movements. She walked badly. Her temperature was 37·8° C. (100° F.) and pulse rate 120 per minute. There was no evidence of endocarditis. The child began to settle down soon after admission. Her speech was bad. The tonsils were unhealthy and they were removed in June, 1935.

In August, 1935, she was not so well. The movements of the upper limbs were very unsteady, especially the left. She had no control of the urine or fæces. There were no abdominal reflexes, but all deep reflexes were active. The heart beat was rapid and the sounds poor in tone. She was somewhat drowsy. The ocular fundi were normal. There was no nystagmus, no paresis of ocular muscles and no loss of reaction to light.

When the child was admitted to hospital there was no excess of hair, but by the end of 1935 she was exceedingly hairy, the hair being most abundant on the back, limbs and cheeks. There was no change in her behaviour. She behaved as a little girl and did not resemble a boy in her behaviour in any way. A skiagram of the pituitary region showed no change, and a urogram showed no alteration in the kidney shadow.

In January, 1936, she shed all her excessive hair, which was found in the bed each morning in large quantities.

About September, 1935, she had had an offensive yellow vaginal discharge, which was copious. No gonococci or trichomonas were found. Many smears were taken, but the pathological report was always the same, namely, a vast mixture of organisms. This discharge was later followed by well-digested greenish motions, with a most objectionable smell. These later became normal.

At the end of February, 1936, the vaginal discharge returned. The discharge was yellow and profuse and later became purple-stained. Still later it became black. The motions were also black and offensive.

Dr. Windsor examined the child under a general anæsthetic. There was a vaginitis extending up to the cervix, which was eroded. There was no intraabdominal swelling.

Microscopic examination of the stools gave no information of importance. Mantoux and Wassermann tests gave no reaction. Lumbar puncture was unsuccessful. The temperature was seldom raised, and then only slightly. The pulse rate was always raised, varying between 90 and 140 per minute.

Dr. Earnshaw considered that the patient was suffering from chorea, involving the cerebellum and pituitary. He said that chorea was really cerebral rheumatism. It was the most frequent single manifestation of rheumatism. Pathologically the condition was one of meningo-encephalitis. At autopsy perivascular infiltration was found with cells, with inflammation of the *pia mater* and toxic changes in the cerebral cells, more especially those of the cortex. The cerebro-spinal fluid pressure might be raised, with sometimes an increase in cells, and occasionally organisms might be found.

The hirsutism made one think of the possibility of a tumour of the adrenal cortex or of the pituitary. No palpable tumour could be felt in the renal regions, and a urogram revealed no distortion of the kidney pelvis. This, however, did not rule out the possibility of an adrenal cortical tumour. An X ray examination of the pituitary fossa revealed no abnormality.

Dr. Earnshaw said that if the hirsutism had been due to a tumour of the adrenal cortex or to a tumour of the pituitary, more particularly of the anterior pituitary, thus causing an obstruction or depression in the flow of the posterior pituitary secretion, the symptoms, such as the hirsutism, would not have cleared up. Whatever caused the hirsutism, it must have been of a temporary nature, and it was reasonable to suppose that the rheumatic encephalitis spread temporarily to the pituitary, just as acute encephalitis might spread to the pituitary and along the optic nerve, for, after all, the posterior pituitary and the optic nerve were really prolongations of the brain substance.

Transposition of Viscera.

Dr. Earnshaw also showed another patient, who was admitted to hospital on account of left apical pneumonia. His recovery was uneventful. The patient had, however, transposition of the thoracic and abdominal viscera. The heart was on the right side, the apex being within and below the right nipple. The liver was on the left side. A barium meal showed that the alimentary tract was also transposed.

Dr. Earnshaw said that such conditions were very rare, but had no influence on the patient's longevity.

Cervical Swelling.

DR. A. LYNCH showed a boy, aged six and a half years, who was born in Brisbane and had always lived there. The father and mother were healthy and there were no other children. Four months ago the child had had pertussis. The mother had noticed a swelling in the neck from birth; there was a huge central lump from the chin to the upper end of the sternum, which, after a few days, decreased to half its original size. There was then a very gradual increase in size until the pertussis appeared four months ago. After that the increase had become rapid. There had been no signs of pressure, no difficulty in swallowing or breathing or any involvement of adjacent nerves or vessels. There had been no signs to suggest a toxic goitre. On examination there was a considerable swelling in the region of the thyreoid, in the mid-line, and on both sides of the neck. The consistency of the swelling was soft, and the shape was irregular. A thrill could be palpated and a soft systolic murmur heard. The mass moved on swallowing. Nothing abnormal was found in any of the systems. There was no exophthalmos and the skin was not unduly warm or moist. Since the child was admitted to hospital on May 2, 1936, the temperature had been normal, the pulse had varied between 90 and 120 per minute, the respirations between 20 and 28 per minute. The bowels had been acting fairly well. The child had been sleeping well and there was some cough. He was being given Lugol's solution, 0·12 cubic centimetre (two minims) in milk three times a day.

Subacute Glomerular Nephritis.

DR. H. MATHEWSON showed a girl, aged ten years, who was admitted to hospital on March 27, 1936. Five weeks previously she had complained of pain in the foot, which was swollen; the pain extended to the groin, then two or three days later it disappeared. The face had then become puffy, the child was put to bed and had been in bed since. She had occasional attacks of vomiting, and was constipated. She had been passing dark smoky urine for three or four weeks, but there was no marked frequency. On her admission to hospital her temperature was 37·2° C. (99° F.), the pulse rate 96 per minute, and the respiration rate 24 per minute. The throat was injected, the tongue was coated with brown fur, and the breath was offensive. There was nothing abnormal in the heart or lungs. There was some tenderness in the loins, but no ascites. The face was puffy and there was generalized œdema. On April 1, 1936, it was reported that the girl was passing between ten and twenty ounces of urine a day, at first acid and then alkaline after the administration of potassium citrate. The urine contained "three-quarters albumin" and blood; the specific gravity was 1010. Microscopically the urine contained some pus and much blood.

On April 7, 1936, the girl was given ten grammes of urea twice a day for two days, then three times daily for two days. The urinary output then increased to the region of 50 to 60 ounces in the twenty-four hours, and the amount of albumin present decreased. The amount of blood also decreased slightly. The patient continued to be given 15 grammes of urea three times a day till April 27, 1936, when it was ceased. The urinary output then decreased to 14 ounces a day. The systolic blood pressure was 130 millimetres of mercury and the diastolic 95 millimetres of mercury. On April 30, 1936, the doses of urea were recommenced, with the same increase in output. The urine was acid in reaction and still contained a cloud of albumin, though there was very much less blood. On May 6, 1936, microscopic examination of the urine revealed many red blood cells and epithelial casts. The blood cholesterol showed 250 milligrammes per 100 cubic centimetres of blood. The œdema had much decreased since the output was increased.

Correspondence.

A CORRECTION.

SIR: My attention has been called to a misleading statement in the "Medical Annual" for 1936. The statement is: "J. C. Storey quotes a fatality rate of 40·83 per cent in 289 cases operated on in Australia for appendicitis with diffuse peritonitis." I desire to make it perfectly clear, in justice to Mr. Storey, that these were not his own personal cases, nor is it certain that all of the 289 were, as a matter of fact, operated on. Mr. Storey's own figures compare very favourably with those published elsewhere (925 cases with 12 deaths, a mortality of 1·29 per cent.).

Yours, etc.,

A. RENDLE SHORT.

Department of Surgery,
Canynge Hall,
Whatley Road,
Bristol 8.
July 15, 1936.

THE LAWRENCE-MOON SYNDROME.

SIR: I was interested to read, in the journal of May 2 last, of a case shown by Dr. E. O. Marks at a meeting of the Queensland Branch of the British Medical Association on February 7.

There seems little doubt that the case is one of the Lawrence-Moon syndrome. Two brothers with this condition were shown at a recent post-graduate course at the National Hospital, Queen Square. These were aged fifteen and twelve. They had been healthy at birth: full-time, large babies. They were both a little slow in walking and talking. The elder had reached standard V at school, and the younger was in standard IV.

The only additional point in the past history was that the younger had, two years previous to being shown, developed head-nodding whenever he fixed his gaze on anything. Since then his vision seemed to have deteriorated progressively.

The older boy's vision went off later and also deteriorated progressively.

The younger had polydactylism (six toes on each foot), had poor vision, was fat, and was described by the demonstrator, Dr. Kinnear Wilson, as being *arrière*.

The elder had had six fingers on the left hand, one having been removed by operation. They both had retinal degeneration without, however, pigmentation, which is not uncommon in this rare condition.

The syndrome was first described about 1866 and redescribed by Bredl in 1922.

Unfortunately I have not these references, but it was stated that the visual defect in these cases is frequently characterized by night blindness and often by *retinitis pigmentosa*.

A considerable proportion are, it is said, dwarfish (? pituitary), but by no means all, and some have been overgrown.

Yours, etc.,

G. C. Moss.

Royal Chest Hospital,
City Road,
London, E.C.1.
July 2, 1936.

HOME FOR INCURABLES AT RYDE, NEW SOUTH WALES.

SIR: The management of "Moorong" home for incurable cancer cases and radium treatment centre is concerned that the home is not being fully utilized, and believes that the reason for such lack of use can only be that the attention of suitable patients and their relatives has not been drawn to the facilities offered.

"Moorong Cancer Home" and the "Radium Treatment Centre" are controlled by the New South Wales Homes for Incurables. The home is situated at Ryde in beautiful surroundings, standing in its own grounds, comprising about 44 acres. Practically all the dairy produce required for the home comes from the home farm, which is on the estate.

The home is run on modern scientific lines and the matron and sisters are all fully qualified. At the "Radium Treatment Centre" all forms of malignancy are treated by our honorary medical staff.

The home receives no government subsidy and the patients are asked to pay only what they can afford.

I should like to feel that all cases coming under the above category have the opportunity of coming to the home, and I believe that the medical profession can be of great assistance to those afflicted by this disease, in advising them of the existence of this home. Full particulars and application forms may be obtained from the Secretary, New South Wales Homes for Incurables, 70-72, King Street, Sydney (G.P.O. Box 1307 J).

Yours, etc.,

ROBERT C. DIXSON,
Chairman of the Board.

Ryde,
July 28, 1936.

"POISON GAS" AS A WEAPON OF WAR.

SIR: We are informed by cable service (Melbourne *Herald*, August 7) that "a plan for the worldwide cooperation of doctors in an effort to prohibit poison gas warfare is being discussed privately by the British Medical Association". The news item states that the British Medical Association in Australia is expecting an approach with a view to cooperation "when the Council has formulated plans"—with the strong inference that such cooperation would be forthcoming.

I suggest that before we commit ourselves to such a campaign we shall do wisely if we examine closely into the matter and apply to it more exactly than has hitherto been the fashion the scientific method of induction from ascertained facts on which rests our claim to be heard on social questions such as this. I submit as among facts relevant to the problem the following:

(1) The military authorities teach that the gas weapon is one of the most humane methods hitherto invented by man for imposing his collective will on his fellows—whether these be a rowdy mob or an opposing national army. So far as the facts at my disposal are a guide, this view seems justified. In the Great War the proportion of deaths was much smaller among battle casualties due to "gas" than in those caused by missiles. And from the point of view of pain, chlorine and other violent irritants, which give a cruel death, were of little value as weapons after the first shock of surprise. Those chiefly used, as most effective against a prepared enemy—mustard gas, phosgene, the lachrymators and the chlorarsenes—were for the most part much less lethal and their effects not more dreadful (commonly far less so) than the wounds inflicted by high explosives, bombs or bullets. The argument that crippling ill-health will later result from the lesions caused by gas—even from "occult" gassing—in greater degree than from other forms of wounding has no support in proved facts.

(2) The moral victory won in the war by Germany, who has imposed on the world her own criteria of "right" and "wrong"—her *cultur*—a victory achieved when the Allies followed her lead into "offensive" gas and retaliatory bombing of undefended towns, would seem to have insured that in "the next war" the cannon fodder, so to speak, will chiefly be sought and found, not in the soldiers, but in civilians, through the bombing and gassing of vital economic and administrative centres. The morale that will count will not be that of the soldier, but of the community as a whole. Now a community can be effectively protected against gas; it cannot be effectively protected against bombing by high explosives.

(3) The efficiency of "gas" as a weapon was largely inhibitory—wearing a gas-mask "cramped the style" of the most ardent fighter—and psychological—a mystery surrounded the lesion caused by it, akin to the concept "shellshock". The efficacy of "gas" is in fact a measure not so much of the physical courage of those attacked as of their "essential guts"—moral courage and intelligence.

There has, indeed, been created in connexion with poison gas a phobia which must be combated by all means in our power as an essential measure of national defence. I would even go so far as to suggest that the British Medical Association should consider whether it should not rather move to restrict all warfare in future to poison gas. Alternatively, if it wish to be of service, it should cooperate in an educational campaign with the purpose of clearing the psychological atmosphere that has been created—not altogether fortuitously—around the subject by many and various influences, but chiefly through those two most potent collaborators in the confusing of thought and stultifying of action—fear and mystery.

If the profession does indeed desire to get its teeth into some great project for the betterment of human relations, by tackling the problem of war not by treatment but by prevention, I suggest that we might take up the matter of tariff barriers. "But", some will say, "here is a crank!" I am unrepentant.

Yours, etc.,

A. G. BUTLER.

Undated.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

THE annual meeting of the Australasian Medical Publishing Company, Limited, was held at The Printing House, Seamer Street, Glebe, New South Wales, on August 24, 1936, Dr. T. W. Lipscomb, the Chairman, in the chair.

Directors' Report.

The Directors' report was presented as follows:

The Directors submit their report for the past year and the balance sheet as at June 30, 1936, together with the profit and loss account for the twelve months ended June 30, 1936.

It is with deep regret that we have to report the death of Dr. William Henry Crago. He was the first chairman of directors of the company, and much of its progress was due to his untiring effort. On July 6, 1936, a bronze tablet was erected at The Printing House to his memory.

THE MEDICAL JOURNAL OF AUSTRALIA retains its position as a scientific publication and maintains its standard. During the year provision was made for the extension of editorial discussions, and a number of special articles were published.

The printing and publishing department continues to turn out a large volume of work, and recently it was found necessary to install another linotype.

The result of the transactions of the company for the twelve months is slightly better than that of the previous year, and provision has been made for the payment of debenture interest for the year ended June 30, 1936.

During the year the sum required for the redemption of debentures registered in the names of deceased debenture-holders has been larger than usual, and there will be a further increase in future years. It is thought that it would be more satisfactory if some of the younger members of the Branches in the several States took over a number of these debentures when they fall due for redemption, rather than that they should all be redeemed by the company. If this is done, it will result in the retention of the present personal interest in the company, of members resident in all States of the Commonwealth.

Dr. D. D. Paton and Dr. Gregory Sprott retire from office by rotation, in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

T. W. LIPSCOMB,
Chairman.

August 24, 1936.

Election of Directors.

Dr. D. D. Paton and Dr. Gregory Sprott were reelected to the Board of Directors.

University Intelligence.

THE UNIVERSITY OF ADELAIDE.

THE sum of one hundred and seventy pounds has been paid to the University of Adelaide by the former pupils of Dr. Archibald Watson, Emeritus Professor of Anatomy, for the purpose of founding a prize in his honour. The Council of the University of Adelaide, on the recommendation of the Faculty of Medicine, has adopted the following statutes:

1. There shall be an annual prize, to be called the "Archibald Watson Prize".

2. The prize shall consist of a printed reproduction of the portrait of Archibald Watson and the sum of six guineas.

3. The prize shall be awarded to the medical student who, after the completion of his term of surgical dressership, shall, upon examination, be found to be the most proficient in applied surgical anatomy.

4. If the examiners shall not consider any candidate worthy of the award the prize shall lapse for that year.

5. The examiners shall be appointed by the Council on the recommendation of the Faculty of Medicine.

6. The prize shall not be awarded twice to the same person.

These statutes may be varied by the Council from time to time, but the title and general purposes of the prize shall not be altered.

The British College of Obstetricians and Gynaecologists.

THE ADMISSION OF MEMBERS.

THE attention of intending applicants is directed to the revised regulations regarding the admission of members to the British College of Obstetricians and Gynæcologists (M.C.O.G.). Candidates, in addition to submitting case records, must now sit for two written papers as well as a clinical examination. Those interested should communicate for fuller details of the new regulations with any member of the Reference Committee for Australia: Professor Windeyer (Sydney), Professor Allan (Melbourne), and Dr. T. G. Wilson (Adelaide).

Obituary.

DANIEL SAMUEL COTO.

WE regret to announce the death of Dr. Daniel Samuel Coto, which occurred on August 10, 1936, at Koroit, Victoria.

ERIC WILLIAM BERESFORD WOODS.

WE regret to announce the death of Dr. Eric William Beresford Woods, which occurred on August 12, 1936, at Melbourne, Victoria.

ROBERT MAXWELL McMASTER.

WE regret to announce the death of Dr. Robert Maxwell McMaster, which occurred on August 23, 1936, at Sydney, New South Wales.

Books Received.

CLINICAL HANDBOOK FOR RESIDENTS, NURSES AND STUDENTS, BEING THE ROUTINE METHODS OF ST. VINCENT'S HOSPITAL, SYDNEY, by Members of the Staff of the Hospital; edited by V. M. Coppleson, Ch.M., F.R.C.S., F.R.A.C.S., and D. Miller, Ch.M., F.R.C.S., F.R.A.C.S.; Second Edition; 1936. Australia: Angus and Robertson Limited. Crown 8vo, pp. 215. Price: 6s. net.

FAMILY MEALS AND CATERING: A COOKERY BOOKLET FOR HOUSEWIVES; 1936. London: The British Medical Association; Australia: Angus and Robertson, Limited. Demy 4to, pp. 27, with illustrations. Price: 9d.

NARRATIVE OF AN INVESTIGATION CONCERNING AN ANCIENT MEDICINAL REMEDY AND ITS MODERN UTILITIES: THE *SYMPHYTUM OFFICINALE* AND ITS CONTAINED ALLANTOIN, by C. J. Macalister, M.D., F.R.C.P., together with an Account of the Chemical Constitution of Allantoin, by A. W. Titherley, D.Sc., Ph.D.; 1936. London: John Bale, Sons and Danielsson, Limited. Crown 8vo, pp. 60. Price: 2s. 6d. net.

MANUAL OF EMERGENCIES, MEDICAL, SURGICAL AND OBSTETRIC: THEIR PATHOLOGY, DIAGNOSIS AND TREATMENT, by J. Snowman, M.D., M.R.C.P.; Third Edition; 1936. London: John Bale, Sons and Danielsson, Limited. Crown 8vo, pp. 410. Price: 10s. 6d. net.

POCKET-MONOGRAPHS ON PRACTICAL MEDICINE: TREATMENT OF FRACTURES IN GENERAL PRACTICE, by W. H. Ogilvie, M.D., M.Ch., F.R.C.S.; Volumes I and II; Second Edition; 1936. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo, pp. 188, with illustrations. Price: 2s. 6d. each volume.

Diary for the Month.

SEPT. 1.—Tasmanian Branch, B.M.A.: Council.
SEPT. 2.—Western Australian Branch, B.M.A.: Council.
SEPT. 3.—Victorian Branch, B.M.A.: Branch.
SEPT. 3.—South Australian Branch, B.M.A.: Council.
SEPT. 4.—Queensland Branch, B.M.A.: Branch.
SEPT. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 9.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 11.—Queensland Branch, B.M.A.: Council.
SEPT. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 15.—Tasmanian Branch, B.M.A.: Council.
SEPT. 16.—Western Australian Branch, B.M.A.: Branch.
SEPT. 17.—New South Wales Branch, B.M.A.: Clinical meeting.
SEPT. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 23.—Victorian Branch, B.M.A.: Council.
SEPT. 24.—South Australian Branch, B.M.A.: Branch.
SEPT. 24.—New South Wales Branch, B.M.A.: Branch.
SEPT. 25.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. S. B. Forgan has been reappointed a Member of the Medical Board, Port Pirie, South Australia, in accordance with the provisions of the *Workmen's Compensation Act*, 1932.

* * *

Dr. C. T. Turner, of Mount Gambier, South Australia, has been appointed Medical Referee for the purposes of the *Workmen's Compensation Act*, 1932.

* * *

Dr. L. W. Jeffries, of Adelaide, South Australia, has been appointed Medical Referee for the purposes of the *Workmen's Compensation Act*, 1932.

* * *

Dr. C. T. Piper has been appointed Deputy Quarantine Officer, Port Lincoln, South Australia, under the provisions of the *Quarantine Act*, 1908-1924.

* * *

Dr. A. S. Grimwade has been appointed Certifying Medical Practitioner at Geelong, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi-xviii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer.
DEPARTMENT OF MENTAL HYGIENE, MELBOURNE, VICTORIA: Medical Officer.
KALGOORLIE DISTRICT HOSPITAL, KALGOORLIE, WESTERN AUSTRALIA: Resident Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.
NEW SOUTH WALES MASONIC HOSPITAL, ASHFIELD: Resident Medical Officer.
SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Instructor in Practical Anæsthetics.
THE PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer, Resident Medical Superintendent.
THE QUEEN'S (MATERNITY) HOME INC., ROSE PARK, SOUTH AUSTRALIA: Resident House Surgeon.
THE UNIVERSITY OF MELBOURNE, VICTORIA: Stewart Lectureship in Medicine.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Temporary Honorary Officers, Chief Resident Medical Officer, Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—23RD YEAR. SYDNEY, SATURDAY, SEPTEMBER 5, 1936. No. 10.

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The E. C. Stirling Lectures.[1]

LECTURE II.

LOSS OF CONSCIOUSNESS OF PRIMARY NEUROGENIC ORIGIN.

By ALLAN S. WALKER, M.D. (Sydney),
Sydney.

ACTUALLY, all suspension of consciousness must be ultimately, even if not originally, due to neurogenic change. The rough definition of unconsciousness which is sufficiently explicit in considering the results of faults in the vascular supply to the brain, cannot be regarded as adequate when we come to discuss defects of consciousness from the cerebral point of view. What is consciousness? Hughlings Jackson, with his clear and philosophic insight into the mechanism of cerebral function, said:

> There is no such entity as consciousness in health; we are from moment to moment differently conscious . . . As the highest nervous arrangements are only highly complex sensori-motor arrangements representing the whole organism, there are degrees of loss of power over the body corresponding to what are psychically degrees of affection of consciousness.
>
> The highest nervous processes are potentially the whole organism.[(1)]

Grades of consciousness must therefore vary according to the cerebral levels affected; Jackson attacked the false concepts which led medical observers into such errors as that of describing a patient as "confused but quite conscious", or of speaking of loss of consciousness as synonymous with coma. If a patient is confused, if he cannot ideate on the highest plane, if his memory and judgement are at fault, he has a defect of consciousness, using the word in its highest and fullest sense.

[1] Delivered at Adelaide, May, 1936.

And from such lesser grades to the major grades in which bodily functions are also abrogated, it is but a question of a step or several steps; the principle remains the same.

And where is the seat of consciousness? Rather, where are the seats of consciousness? We can, as Jackson pointed out, indicate the anatomical substrata of consciousness without in any sense attempting an explanation of consciousness itself, and such substrata are not only multiple, but constantly changing with our changing adjustment to our environment. Consciousness, then, is an even more generalized expression of cerebral activity than intelligence, and in speaking of its loss we must clearly state from time to time and from case to case exactly what we mean. There is, of course, a localization of each component of consciousness, but although we regard most of these components as belonging to the higher order of nervous processes, we cannot locate them either in one place or even at one neural level. I lay some stress on this, for we still suffer to some extent from certain exaggerated attempts to pigeon-hole cerebral functions, an error which is exposed if we try to push it to its ultimate conclusion; the last absurdity in postulating "centres" for specialized acts would be to imagine a centre for shaving, or for lighting a cigarette. As Fulton[2] says, we cannot consider higher intellectual activities in connexion with conventional ideas of localization of function.

It is obvious, then, that loss of consciousness cannot be placed in strictly limited categories, but certain rough subdivisions are useful; as indicating certain states in which normal full consciousness is lost or disturbed.

(1) Sleep.
  (a) Physiological.
  (b) Pathological: (i) Due to a definite nervous lesion, (ii) due to no demonstrable lesion.

(2) Complete loss of consciousness other than sleep.
  (a) Due directly or indirectly to an irritative, disruptive or destructive lesion.
  (b) Due to no demonstrable lesion.

(3 Lesser disturbances of consciousness.
  (a) Partial loss of consciousness.
  (b) Aberrations of consciousness.

It is necessary first to point out that sleep differs in nature from other forms of loss of conscious cortical control: not only is the phenomenon quite distinct in itself, but it is produced by a different mechanism. Sleep is an impressive phenomenon—a nightly or daily recurring state in which the whole parasympathetic apparatus is dominant, in which even the deep reflexes may be in abeyance, and in which even the ever-watchful respiratory centre becomes less sensitive to carbon dioxide.[3] Its nature has been somewhat clarified by the physiological studies of Pavlov on conditioned reflexes, by pharmacological investigation into the mode of working of hypnotic drugs, and by study of certain cerebral tumours which give rise to pathological grades of sleep. Only the briefest mention of these can be made. Pavlov showed that a stimulus which at first aroused the cortex to action, if constantly repeated, produced the opposite effect through a process of inhibition as it were spreading over the cortical areas. Thus we can explain the power of monotony, of circulatory and other metabolic variations and certain drugs to produce sleep, associated with or apart from fatigue; in these terms the impulse to sleep starts from the cortex. But tumours and other lesions in the region of the floor of the third ventricle are known to have the power of causing hypersomnia. It is of interest in passing to recall that Oliver, of Bath, in 1709 recorded notes of a case of pathological sleep.

This condition is different from that of the general torpor due to increased intracranial pressure, for the patient may be aroused as from normal sleep. Now herein lies the essential distinction between sleep, however abnormal in degree and periodicity, and other forms of loss of consciousness. Sleep is reversible, and that

> gentle thing
> Beloved from pole to pole

may be abolished by any simple physical interruption, whereas other neural types of unconsciousness are irreversible: the state of consciousness cannot be restored at the will of the observer.

That there is a sleep regulating centre indeed seems an almost inevitable conclusion on studying the work of modern neurologists, but, as von Economo[4] points out, its action probably consists in a coordination of the different changes occurring in our vegetative anatomical and psychological system. No doubt this function is deeply rooted in the vegetative nervous system, and if we conceive that in the operation of the sleep centre the cerebral centres of the higher cortical and thalamic orders are inhibited, its action becomes intelligible. External influences may slacken the reins in the hands of these higher centres, or internal influences such as fatigue and monotony *et cetera*. Further, it is possible to see how a reversible action may occur by an exertion of higher frontal cortical control. It is a matter of experience that both mental effort and an appreciation of sensation of the order we may call thalamic may defeat an otherwise almost overpowering urge to sleep, that demon who seeks to overwhelm us in awkward situations, even during technical lectures. Syncopal and narcotic loss of consciousness are obviously of a different order to normal sleep, however temporarily it may be that they "slit the thin spun life"; their action cannot be reversed until the cause is removed.

It is a fascinating hypothesis that "hypnotoxins" may be produced which may act directly on the sleep centre; and Zondek and Bier would work the hard working pituitary gland still harder by their claim that a "brom-hormone" is produced by it. Claparède's aphorism is that "we sleep, not because we are intoxicated by hypnotoxins, but in order not to get intoxicated by them". Remembering the great restorative effect of "sore nature's bath, balm of hurt minds", this saying seems to penetrate nearly to the truth of the matter.

The causes of sleep aberrations are several. There may be a definite lesion, usually a cerebral tumour. A recently observed example is as follows:

A woman, aged fifty years, had suffered for several months from headache, loss of power of concentration and what she termed "giddiness". The last-mentioned symptom was found to be a more or less gross incoordination of the right leg, causing staggering; the right arm was also incoordinate and subject to frequent athetoid movements, and myoclonic twitchings were observed in the muscles of the right leg. Two weeks later some lack of control was noticed in the left leg also. Following these the athetoid and myoclonic movements ceased and she became drowsy.

This state of drowsiness was one of pathological sleep, since she slept peacefully through much of the day, but could be roused without trouble, when she rubbed her eyes and smiled and was then prepared to talk or take food. Soon her mental condition became less alert even when aroused, her powers of thought and concentration dwindled, and at this stage papilloedema appeared for the first time in both eyes. Subsequently at operation a cyst was located deep in the posterior part of the left frontal lobe.

Undoubtedly this tumour had affected not only the basal gangliar region, but also the zone in the floor of the third ventricle. An important point in this case history is that at the time when these notes were originally written the patient, though showing early signs of intracranial pressure, suffered only from slight hebetude. It was the constant falling asleep that was the notable feature, due possibly to release of the sleep centre from its normal control by other higher centres.

In other cases no demonstrable lesion has been found; such are the cases of the so-called "idiopathic narcolepsy", though I shall not attempt to touch on the possible neural or endocrine factors which may be related to this condition. In this strange malady the manifestations are much the same whether they follow a known disease such as encephalitis, or form an association with the still unexplained epilepsies. A contrast of the two leading features of narcolepsy leads us to some interesting considerations. These features are firstly, attacks of uncontrollable sleep occurring in the most inconvenient, unexpected, or even grotesque circumstances, and secondly, the so-called "cataplectic" attacks, in which the patient is suddenly rendered helpless, though not unconscious, toneless, inert, and unable to move, usually as the sequel of some powerful emotion.

Thus in narcolepsy we have an inhibition of control, but not necessarily of consciousness; that is, control of muscular coordination and of the vegetative sleep centre is lost. It would seem that the active centres involved in setting free these particular release mechanisms are in the region of the hypothalamus; they are of particular interest as illustrating the effects of loss of control of the brain at one particular level. It will be seen that both these happenings are only exaggerations of the normal: we have all fallen asleep when we should have remained awake, even, for instance, when driving a motor car; we have all been helpless for brief periods when under the temporary dominance of strong emotion, such as laughter. Kipling describes the latter aptly in "The Puzzler".

He waggled his hands at us, and when we entered we saw that the man was stricken speechless. His eyes grew red—red like a ferret's—and what little breath he had whistled shrilly. At first we thought it was a fit, and then we saw that it was mirth—the inopportune mirth of the Artistic Temperament.

Perhaps the same mechanism is at work in producing some of the phenomena of hysteria, for example those occurring under the influence of emotional exaltation. We may recall the power of certain monks of mediæval times who could induce a trance of hypnotic type in themselves by the rapt contemplation of a fixed object. In a lesser degree the same end may be attained even by the more worldly, who may experience some emotional dislocation of higher consciousness, so that they feel, like a modern imaginative heroine of fiction, that "for a moment body and spirit were harmoniously one, floating in a pure eddy of time".

True or Irreversible Loss of Consciousness.

In the category of true or irreversible loss of consciousness must be placed many serious and important diseases of the nervous system; inflammatory diseases such as encephalitis, meningitis and syphilis should perhaps be placed in the vascular series, but in any case they are of great importance, particularly the great imitator, syphilis. Intracranial tumours and trauma are other lesions that are of peculiar interest, especially as their results may closely parallel those produced by more or less severe vascular lesions such as hæmorrhages or the hypertensive encephalopathies. The significance of loss of consciousness of all grades as a feature of concussion or the more chronic cerebral contusion should be noted; it is strange to reflect that a mere shake of the head, so to speak, may abolish conscious life so completely for a time. Lastly we come to that remarkable series of conditions that are symptomatically grouped as the epilepsies. Infantile convulsions may also be included here; the phenomena are similar, there is often a plausible reason for them, and perhaps, too, a corresponding basis in the nervous system. This wide group of epilepsies includes, symptomatically at least, a number of the lesions just mentioned, and to this we now turn our attention.

The major epileptic attack may be taken as the starting point. Practically the first and the most enduring symptom is loss of consciousness, which is usually complete. There is always, in addition, an aftermath of disturbed consciousness, a period during which the patient is confused or stuporose, or may display some of those curious post epileptic phenomena to which I shall presently refer. Occasionally the loss of consciousness may be severe and prolonged to the point of definite physical danger, in the perilous *status epilepticus*. It may be pointed out as an interesting fact that loss of sphincter control is not necessarily a part of the epileptic attack; while it may follow or accompany slight seizures, it may sometimes not be associated with major attacks. The same may be said in some degree of tumours of the frontal lobe.

Loss of consciousness in an epileptic seizure is not necessarily due to the severity of a fit, it is more a question of the level involved, and the spread from level to level. Some important information has been gained by studying the seizures arising from frontal lobe lesions. Penfield and Gage[5] point out that these fits are stigmatized by loss of consciousness,

often without any aura, together with turning of the head and eyes to the opposite side, and a simultaneous convulsion of the opposite extremities. When the seat of the attack is in the pre-central or post-central gyrus, or behind the central sulcus, consciousness is usually lost late. In the localized Jacksonian attacks it is significant how loss of consciousness will occur if there is sufficient spread of the epileptic neural discharge. The light shed upon curious symptoms by loss of consciousness is shown in the following case history.

A man, aged thirty-five years, complained for several months of strange spasmodic twitches of the face and jaw. Latterly these had been more severe, and his jaw and then his head seemed forcibly turned, as though they were dominated by some compulsive force which he could not control. These attacks aroused the mirth of his fellow-workers, until one day their mirth changed to alarm when during such a spasm he fell unconscious to the floor. At operation a localized tumour was found in the Rolandic cortex.

Of course, while recognizing the importance of the frontal lobes in the regulating of the higher conscious functions, we must not lay too much stress on the symptoms occurring in irritative frontal lesions. As Collier[6] has truly pointed out of any lesion within the brain, an organic irritative focus may produce any kind of epileptic seizure from *petit mal* to the *status epilepticus*. He believed also that the lesion was only one factor in the causation of fits; that it was not *per se* epileptogenous, but that the appropriate trigger could fire the gun loaded by the appropriate metabolic conditions.

Akin to the motor Jacksonian attack, which may or may not lead to loss of consciousness, is the seizure in which vasomotor or other autonomic phenomena are prominent. Penfield[7] describes, under the caption of "Diencephalic Autonomic Epilepsy", a case in which there were recurring seizures of curious nature. The patient flushed, sweated, salivated and shed tears, the pupils were dilated, respirations were slow, with some hiccup and Cheyne Stokes breathing; later he became pale, the pulse slowed, and he shivered; occasionally he lost consciousness. A tumour was found in the hypothalamic region.

The following case shows, also, how the motor and autonomic functions are marked out distinctly from those presiding over consciousness in the symptomatology of epilepsy.

A man of sixty years had suffered from dizzy attacks for two or three years. His face would flush, he would feel giddy and have an uncomfortable sensation of being incapable of doing ordinary things. These attacks became more severe. There was a lingual aura, followed by some nausea, dimness of vision and staggering; he paled and sweated, and occasionally vomited. Only in two of the more severe had he lost consciousness. Later a severe attack occurred; he had convulsions, became quite comatose and died, evidently from a tumour in which it seems possible that some vascular change or accident may have occurred.

It is perhaps not necessary to point out that epileptiform fits may be caused by a cerebral lesion such as meningeal adhesions or a tumour or any of the forms of cerebral syphilis which are indistinguishable from those where no focal lesion exists.

It is well known that it is risky to diagnose idiopathic epilepsy in a patient over forty; to a certain extent it is risky at any age: we must always keep in mind the possibility of a serious lesion such as a syphilitic lesion or a tumour, of which some disturbance of consciousness may be the first sign.

One form of epilepsy which demands special attention is the nocturnal variety. This may remain undiscovered for years, and sometimes when the patient's relatives bring him for attention it is seen that they are even more alarmed than if the seizures occurred during the day. But in spite of the chill forebodings of the midnight hours, the outlook of nocturnal epilepsy is as a rule good, and its treatment is satisfactory and simple, for no uncomfortable side effects should follow the administration of the appropriate sedatives. Incidentally the time of dosage in all forms of epilepsy is important, and it is necessary in all cases to determine as far as it is possible the hours at which the attacks are prone to recur. The occurrence of epileptic seizures during sleep raises an interesting point. Can the patient be said to lose consciousness or, rather, to become more deeply unconscious when he is actually asleep? The answer is certainly yes. If consciousness were not lost, it is incredible that such stirring events could happen in his body without awakening the subject of the fit; moreover, definite injury is sometimes inflicted, and on awakening the patient not only has amnesia as regards the drama in which he was the chief actor, but he often feels dazed and fatigued for sometime afterwards. These facts are almost enough of themselves to form a basis for diagnosis in cases in which the patient has been heard to utter strange sounds, even though he has not been actually seen in a convulsion.

But what is perhaps the most interesting of all epileptic phenomena in the present inquiry is the minor attack. The common forms of *petit mal* are familiar to all, but they vary: sometimes the patient may lose muscular control, sometimes he may merely have a temporary suspension of mental activity. Quite the most remarkable of these attacks are those varieties such as described in the following case.

A mother brought a bright little girl of eleven years with the history that she had suddenly started to have numerous strange "turns", in which she became limp and motionless, stared, made no response to sound or touch, and then rapidly recovered. Her face did not change colour and she very seldom fell, and usually continued to hold whatever was in her hands at the time. There was no relaxation of the sphincters during the attacks, which were very brief, lasting a few seconds only. Ten attacks or more might occur daily, but they did not appear to disrupt the child's life. No abnormality was found on examination of this intelligent girl. I gave the usual sedative drugs, but no improvement followed. The mother then returned alone and confided to me that she herself had taken similar attacks as a child and they had ceased at puberty, which she thought might soon arrive in the case of her daughter. She asked if the girl would follow the same course. At that time I had never heard of pyknolepsy, but a few years afterwards found that this case tallied exactly with those so well described by Adie.[8]

The explosive onset, the frequent attacks unaffected by treatment, their invariable and slight nature were characteristic. Later on the remaining essential feature was added, for the fits ceased entirely.

These seizures are most illuminating, they are slight, they are identical, they do not affect muscular coordination or control to any degree; they disturb consciousness only at one level, being apparently a

ILLUSTRATIONS TO THE ARTICLE BY DR. C. H. FITTS.

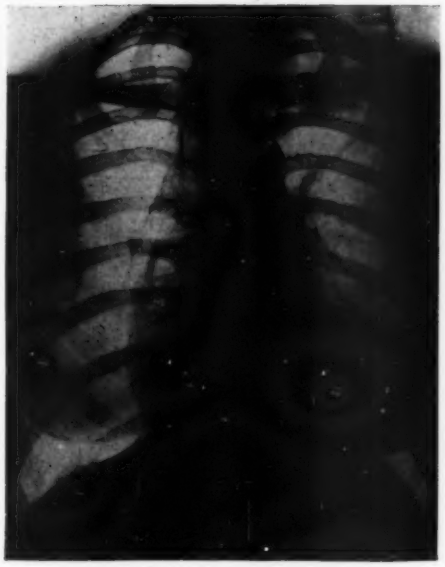
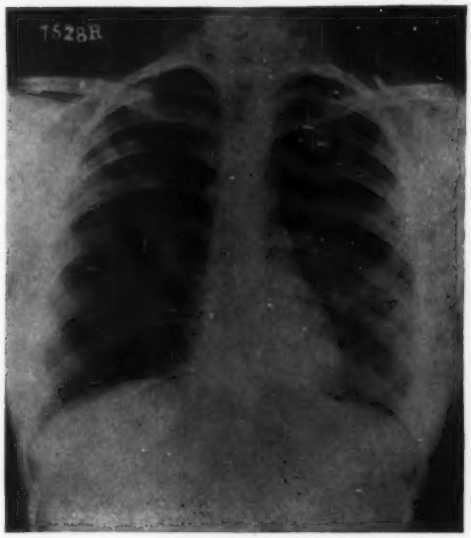


FIGURE I.



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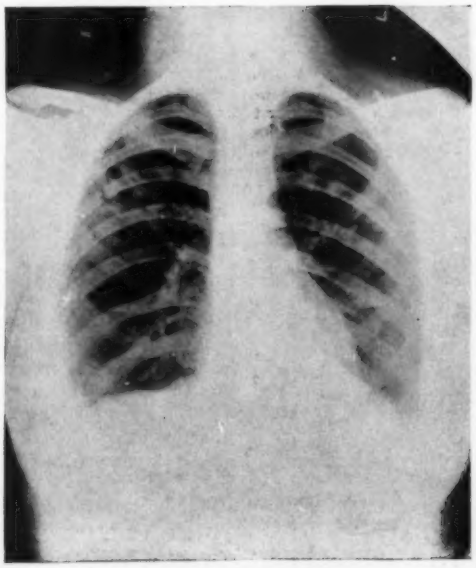

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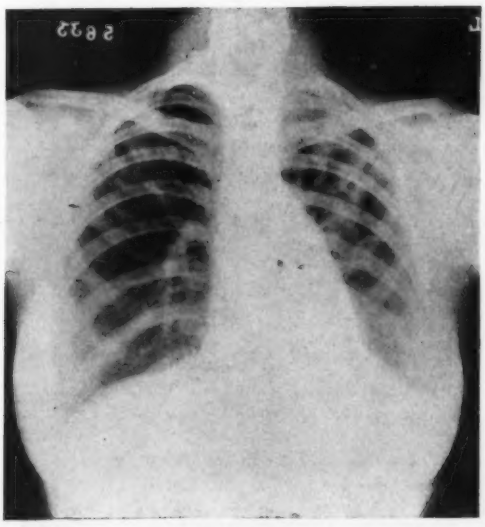

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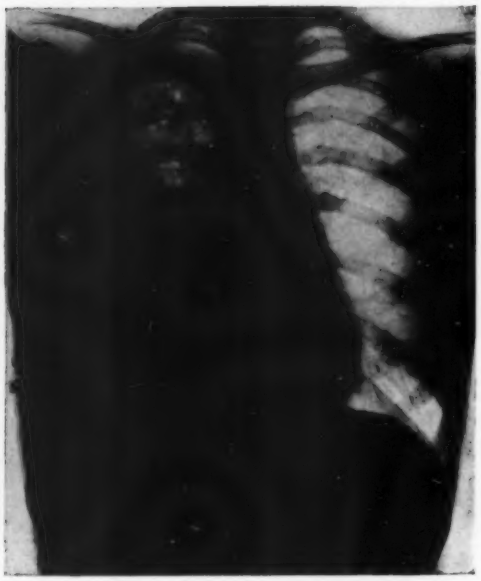
ILLUSTRATIONS TO THE ARTICLE BY DR. C. H. FITTS.



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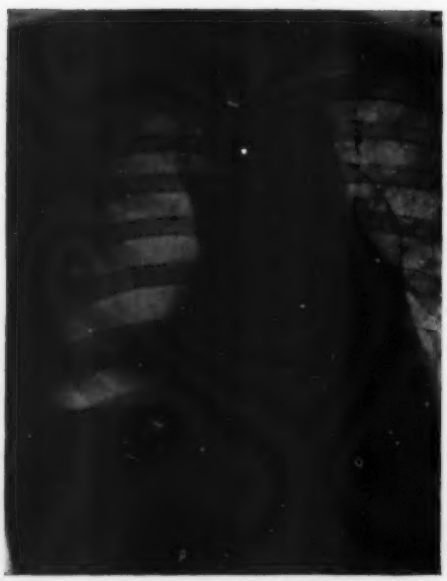

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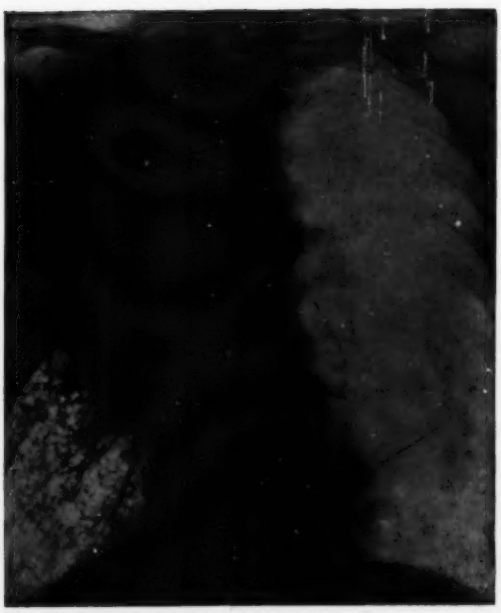

FIGURE VIII.

peculiar refined form of *petit mal*. It would seem possible that some endocrine factor is involved in these cases, since the effect of puberty seems to be beneficial.

It is important to remember that a patient may be unaware that he has attacks of *petit mal*.

Some time ago I was taking the history of a patient who came to me to seek advice as to the correct dosage of sedatives which he was taking to control occasional convulsive fits. I inquired if he had had any minor attacks; he assured me that he had never had any, and had never experienced any temporary confusion or loss of memory. "Have any of your family ever told you that you have stopped speaking in the middle of a sentence or that you seemed to be absent-minded?" I asked. He gazed fixedly in front of him, paled a little, swayed slightly, and after a few seconds of immobility came to himself. "Have they?" I repeated. "What was that, doctor?" he asked. He was even then unaware that he had been through a minor epileptic attack.

It is convenient to restate more or less in order the remarkable grades of severity in loss of consciousness in epilepsy. First there is the *status epilepticus* in which the condition may be prolonged to a quite extraordinary degree. Next comes the major fit after which stupor may persist for a considerable time; all degrees are observed until we come down to the *petit mal* in which the loss is considerable but brief, and pyknolepsy in which it is not only extremely brief but relatively slight, and the one and only symptom. Jackson used to lay stress on the widespread nature of the cerebral change in *petit mal*; he pointed out the initial pallor of the face and the frequency of a slight wave of bodily movement. This is very interesting in view of the possible connexion between vasoconstriction in the face and in the brain occurring in the variety of carotid sinus syncope described previously. Also, there is the fact commented upon by a number of authors, that the mental change following repeated attacks of *petit mal* may be much greater than that seen after many attacks of a severe convulsive nature. Yet in pyknolepsy the prognosis is good, and no sequels have been observed. With this exception, then, we must recognize that the degree of loss of consciousness is not necessarily a measure of the seriousness of the lesion, if any, or of the damage likely to be done.

Still lower in the scale of epileptiform attacks in which consciousness is lost come Jacksonian epilepsy and the curious autonomic variety; in these consciousness may be almost or quite unimpaired.

In the last category of those cases in which loss of consciousness is unusual we may perhaps include vertigo. Symonds[9] agrees with Gowers that consciousness may be lost during an attack of labyrinthine vertigo.

He described an interesting case in which a man of fifty-six years who had suffered from migraine had attacks of tinnitus and deafness, in certain of which he would lose his mental orientation for a moment or two, and would wonder what was happening. Then the patient would recollect that all this was really an incident in a dream that he must have had at some previous time.

This curious feeling of unreality is a characteristic of some epileptic attacks, but it also occurs in migraine, as I now wish to point out.

Thus to the above degrees of disturbances of consciousness we must add still another category, and if I lay what seems undue emphasis on the slight variations from the normal, it is because they are illuminating and may fall within the experience of most of us.

Lesser Disturbances of Consciousness.

I have not attempted to assemble the various degrees of disturbance of consciousness into strict categories; the momentary or partial loss of consciousness occurring in *petit mal* has already been referred to. The suppression of conscious appreciation and attention and other mental disturbances seen in psychiatic practice, such as the cognition defects in melancholia, I cannot deal with here. I wish to refer briefly to certain deranged and incomplete forms of consciousness; the first includes the phenomena following epileptic fits, and the second varieties less important and indeed trivial, but one of considerable interest.

The post-epileptic phenomena shed a most interesting light upon what Jackson called the "anatomical substrata of consciousness" and upon the great importance of recognizing that these must exist at different levels, just as consciousness itself represents an awareness of all manner of physical and psychical activities.

Consider, for instance, the remarkable post-epileptic fugues, such as the following instance.

A young man left his *fiancée* outside a cinema while he went to purchase tickets. He had a slight epileptic attack, followed by some temporary confusion; during this period he wandered off, took a train to a distant suburb, and spent over two days away from home. His actions were apparently quite rational of themselves, though when he came to himself later he had complete amnesia of all that had happened after he left the girl waiting in the street.

The automatism that may follow even a mild attack of *petit mal* is admirably illustrated by Jackson's case of an epileptic medical man who had an attack while he was waiting for a patient to undress. Later he woke to find himself talking to another patient, but in spite of his complete amnesia of the remainder of the previous interview, he found later that he had made a correct diagnosis of a left basal pneumonia and had sent the patient to bed.

Instances such as these must impress us with the truth of the aphorism I quoted at the beginning: that there is no such entity as consciousness. Indeed, allowing for the influence of the old superstition of demonic possession, Karshish, the fictional Arab physician of Robert Browning, described certain of the post-epileptic phenomena clearly.

> The evil thing out-breaking all at once
> Left the man whole and sound of body indeed,
> But, flinging, so to speak, life's gates too wide,
> Making a clear house of it too suddenly,
> The first conceit that entered pleased to write
> Whatever it was minded on the wall.

The other types of disordered consciousness to which I now turn are somewhat more vague. Let us start from the presumably normal and go on thence to conditions of admitted divergence from normal.

Who has not known those curious feelings such as a sensation of unreality or of walking in a dream? And in what circumstances? Usually none other

than the operation of some powerful emotional stimulus which has presumably caused temporary inhibition of the full measure of cortical control. Do these not resemble the more definite sensations often described by patients as that of "floating away"? A veil seems drawn across the brightly lit stage of cerebral activity, the world becomes momentarily unsubstantial, perhaps some threat of greyness and age assails the gay life of normality, as when in the *Nibelung* legend the goddess of youth departed. Or else there is an illusion as of time standing still or of stretching out indefinitely as in the visionary description of Rupert Brooke.

> lifted clear and still and strange
> From the dark woven flow of change
> Under a vast and starless sky
> I saw the immortal moment lie . . .
> . . . . .
> I above time, Oh blind! could see
> In witless immortality . . .
> . . . . .
> You never knew that I had gone
> A million miles away and stayed
> A million years.

As a medical parallel to this I may quote the case of a woman who was given just sufficient ethyl chloride to enable a surgeon to remove a gauze packing painlessly. She was unconscious only for a matter of seconds, but on opening her eyes declared that she had in imagination made a long trip into the country, had walked down a street, met some friends, and conversed with them.

The description of such an illusion is often passed over as mere fancy, as sometimes, indeed, it is, for the complete self absorption of the neurasthenic patient may dull the sensorium so that it cannot perceive the bright colours of the external and objective world of action. It is not always wholly fancy, however, as we shall presently see. Another curious variant of this phenomenon, and one which is not specially associated with any emotional disturbance, is a sensation well described by Oliver Wendell Holmes, who, you will remember, was a professor of anatomy as well as a distinguished author. He speaks of "a sensation that we have been in the same precise circumstances as at the present instant once or many times before". He points out that the circumstances are often very trivial and not such as would fix themselves in the memory, that the impression is very evanescent, that there is a disinclination to record the circumstances or a sense of incapacity to express the state of mind in words, that there is a sense of familiarity about the whole experience, and that it may occur in dreams. It is most interesting to know that Jackson's epileptic doctor-patient made special mention of a curious feeling that in many of his attacks of *petit mal* he was conscious that:

> The central feature has been mental and has been a feeling of recollection, that is of realising that what is occupying the attention is what has occupied it before, and indeed has been familiar, but has been for a time forgotten, and now is recovered with a slight sense of satisfaction as if it had been sought for.

It will be seen how impossible it is to draw a sharp line of distinction in these matters between the normal or physiological and the abnormal or pathological.

Coming from *petit mal* to migraine, we can now link up the curious "distant" feeling familiar probably to all of us with a more definite and quite disturbing sensation of "floating away" or unreality experienced at times by sufferers from migraine. An instance is as follows:

> A young man of twenty-nine years complained of attacks of headache which were heralded by blurring of the central field of vision and restriction of the outer fields. He also suffered from nightmares, from which he awoke sweating and twitching, and with a sensation of what he called "jumping in the stomach". Sometimes when an attack was threatening, following the ocular symptoms, and sometimes at other unrelated times, he would experience a feeling of unreality, as though he were floating away, and had the foreboding that he might become unconscious.

Surely such a history is significant. It would appear that the unreal feeling is actually a disturbance of the function of consciousness, and adumbrates its possible loss. That this causes some alarm to the patient is undoubted, for it is interesting to notice how this story may be given spontaneously, as it was in this case, as one of the outstanding features of the patient's complaint for which he seeks relief.

We may now summarize the position as follows: Consciousness may be lost in sleep, and is then reversible. It may be lost otherwise then by a process of falling asleep, and is then irreversible. Further, loss of consciousness may vary from the deepest coma which is akin to death, of whose loneliness it is too often the forerunner, to the slightest and most transient of subjective sensations. The question is partly one of degree of affection, whether this be by ischæmia, toxic irritation or other unknown cause; it is also partly one of the anatomical level or levels involved. This concept is most important in epilepsy, for it helps us to understand why the same drug should be useful to control convulsions, to prevent minor lapses of consciousness, and even to rescue the subject of *status epilepticus* from his coma. In pyknolepsy the question of sedative drugs is of particular interest, for they are quite unavailing; the condition proceeds quite unaffected by treatment, yet subsides spontaneously. What is the reason for this? Is it in any way related to the apparent absence of the convulsant element in pyknolepsy, and is there something as yet unsupplied by the drugs at present available? This is pure speculation, for we do not know. Indeed we must admit that even apart from attempting to understand the mystery of consciousness we have a vast amount yet to learn about the conditions in which it is impaired or lost.

I have purposely dwelt in some detail upon the lesser aberrations and defects of consciousness in order to stimulate a study of them. Not only are the mechanisms involved of great interest, but a careful consideration of these symptoms may be of the utmost importance to the patient. It is not enough to say that he had a faint or a fit; we should try to find the cause. Are we interested in the patient who has had perhaps only a few minor seizures? We should remember that these may be

due to a tumour causing few if any other signs. Or is that absentminded and seemingly stuporose state functional only, or an early sign of cerebral syphilis? And that strange apoplectiform fit from which the patient so quickly recovered, may it be due to general paresis? In this last instance I have known a whole family accept placidly the gross changes in temperament of the head of the household until such an event occurred. Surely it is not necessary to lay any more emphasis upon the importance of making that earnest study of the individual patient's symptoms and problems which will enable us to apply our knowledge of internal medicine. For though we are now in an era of the scientific side of medicine, in which we essay quite properly, if not always exactly or even discreetly, to measure everything, let us remember that the day of the accurate clinical observer has not closed, nor should it ever close. It takes long experience, great patience, and an equal amount of sympathy and understanding, as well as medical knowledge, to take a history correctly, to evaluate its elements justly, and to trace clearly the march of symptoms. Fortunately for medicine the capacity to do this is possessed by most sound general practitioners. But we must do more than listen and observe: we must keep fresh the spirit of inquiry which ever puts the questions "why is this?" and "what does it mean?" We cannot always answer these questions, but as humble practising physiologists and pathologists we should try.

In Anatole France's mystic and striking romance "Thais" he describes how the unfortunate and much tempted monk, Paphnutius, was wrung by remorse and tortured by desire, and at last, believing that he heard a voice dooming him to spiritual death he "fell lifeless as though struck by lightning". Later he woke to find monks in black robes restoring him. "Without doubt", they said, "the demons had knocked you down and fled at our approach."

Some of these demons responsible for fits or faints are known to us, some we can exorcise, but it may still seem, even to us in the twentieth century, that others have fled at our approach, though our knowledge is so much fuller and our outlook so different to that of the black-robed ascetics. If, however, we too indulge in assumption and speculation, let us at least make them preludes to inquiry.

References.

(1) "Selected Writings of John Hughlings Jackson", Volume I, 1931.

(2) J. F. Fulton: "Some Functions of the Cerebral Cortex", *The Journal of the Michigan State Medical Society*, Volume XXXIII, May, 1934, page 235.

(3) W. R. Hess: "The Autonomic Nervous System", *The Lancet*, December 10, 1932, page 1259.

(4) C. von Economo: "Sleep as a Problem of Localisation", *The Journal of Nervous and Mental Diseases*, Volume LXXI, March, 1930, page 249.

(5) W. Penfield and Lyle Gage: "The Cerebral Localisation of Epileptic Manifestations", *Archives of Neurology and Psychiatry*, Volume XXX, October, 1933, page 709.

(6) J. Collier: "The Lumleian Lectures in Epilepsy", *The Lancet*, March 24, 1928, page 587; March 31, 1928, page 642; April 7, 1928, page 687.

(7) W. Penfield: "Diencephalic Autonomic Epilepsy", *Archives of Neurology and Psychiatry*, Volume XXII, August, 1929, page 358.

(8) W. J. Adie: "Narcolepsy, a form of Epilepsy in Children with a good Prognosis", *The Proceedings of the Royal Society of Medicine*, Volume XVII, 1923-1924, Section of Neurology, page 19.

(9) C. P. Symonds: "The Clinical Significance of Vertigo", *The Lancet*, October 28, 1933, page 959.

OBSERVATIONS ON PHYSICAL SIGNS IN DISEASES OF THE CHEST.[1]

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YOU may wonder how I reconcile some of the observations that follow, with physical signs as we are used to apply them in diseases of the chest, and why I introduce Hippocrates, Galen and others into such a subject. It is because I think it is both interesting and instructive to see how our forerunners dealt with problems which, up to a point, were the same as ours—how at times they found the truth of the matter, at times narrowly missed it, and all too frequently were altogether wrong. When we come to our own times, I hope that it will become apparent that, in the light of our knowledge, we should be discarding those signs which will not stand the test, and readjusting the status of others of which the values have been wrongly assessed. It may appear, from what I have to say, that I subscribe to the opinion that physical signs are valueless. I should like to dissociate myself from that view, and to say that, used with full knowledge of their weaknesses, they are very helpful.

Definition of Physical Signs.

Now I must explain that I am not content that physical signs in diseases of the chest should be confined to inspection, palpation, percussion and auscultation. I think the following quotation from "The Golden Bough" embodies what I would include in a definition of physical signs. It has, too, the merit of appreciating that some physical signs have been recognized since earliest times.

> From the earliest times man has been engaged in a search for general rules whereby to turn the order of natural phenomena to his own advantage. These general rules he deduced after repeated observations. His deductions were true or false. The true or golden rules constitute the body of applied science: the false are magic . . . the bastard sister of science.

The synonym for signs then is natural phenomena, and "natural" here is applied in its broad, though legitimate, sense, and includes phenomena in both health and disease. Observation and true deduction in so far as we are able, in whatever age we live, are the other significant words in this quotation. These are the bases of what Lewis calls clinical science—the study of disease by observation and intimate contact with it, as it exists in living man. It is noteworthy that Lewis himself states that there is practically no problem which he has tackled in the last twenty-five years which does not owe its inspiration to contact with the patient.

The Evolution and Application of Physical Signs from Hippocrates to Laennec.

The seeds of science are curiosity and wonder—two attributes of the unsophisticated mind. The fine flower cannot come until we know all, and that,

[1] Read at a meeting of the Victorian Branch of the British Medical Association on June 3, 1936.

according to that robust optimist Robert Browning, is not in this life. That is why through the last two thousand years we read of the "healing art", and not of the science of healing. Let your fancy roam back to 400 B.C. to the time when the first seed was planted. Imagine yourself born into a world where the healing art was in thrall to religion and magic, with a thousand years of tradition behind it. This was what Hippocrates found. "He brought to it a flexible critical well-poised attitude of mind ever on the look out for sources of error, which is the very essence of the scientific spirit." It is significant, perhaps, that he was contemporary with Sophocles and Euripides, Socrates and Plato, Herodotus and Phidias—men whose works Pericles prophesied would be woven into the stuff of other men's lives. Hippocrates stands for the fundamental importance of seeing clearly, that is, for clinical observation. In the first and third books of "Epidemics" will be found descriptions of two patients with Cheyne-Stokes respiration. Indeed much of his observation was directed to the study of diseases of the chest.

To what use did he put his self-acquired knowledge and his collected facts? Not so much to treatment, for he believed in the *vis medicatrix naturæ*, but to prognosis. His description of the Hippocratic facies comes in the prognostic. With the minutiæ of diagnosis he was not much concerned, and it is doubtful how far he recognized separate disease entities. Prognosis so influenced his writings that they were described by Æsclipiades, who introduced Greek medicine to Rome in the first century B.C., as a meditation upon death. The latter was the first extant fashionable physician, remembered only by the scorn heaped upon him by Galen in his writings. Like his master, Hippocrates, whom he called divine, Galen had an inquiring mind, and attached importance to clinical observation. The efficient healer, he said, must be versed in the three branches of philosophy—logic, the science of how to think; physics, the science of nature in its broadest sense; and ethics, the science of what to do. Unfortunately he was a facile theorist and systematist whose baneful influence did not wane for the next thousand years.

With the Renaissance came the first great anatomists, and chief of these, Vesalius. In the seventeenth century flourished Malpighi, a practising physician, a professor of anatomy, and the author of a treatise on the structure of the lungs. When bidding farewell to his favourite pupil, Vallisneri, he advised him to study Nature and to communicate matters of fact. "Systems", he said, "are ideal, and mutable. Observation and experience are solid and unchangeable". These men, Vesalius the anatomist, and Malpighi the physiologist, I mention because they laid the foundation stones of their respective sciences, and built up the reputation of the Academy of Sciences at Bologna. Over the gateway of this edifice is the following inscription: "The Academy of Art and Sciences of Bologna, for the general use of the whole world."

At the beginning of the eighteenth century, Morgagni came to Bologna, to study there under Valsalva. He made a deep impression, and after a short period of foreign travel he was appointed at the age of twenty-nine, to the Chair of the Theory of Medicine at Padua. Well versed in the traditions and teachings of the greatest medical school of the time, he turned his attention to the correlation of symptoms and signs during life, with the appearance of the organs after death. He laid the foundation of morbid anatomy, and in his eightieth year published "The Seats and Causes of Disease Investigated by Anatomy". These were strange letters to be written to a lay friend, but we must be grateful to the latter for extracting a promise that they should be published unaltered. In this book Morgagni says:

> Those who have dissected many bodies have at least learned to doubt; when others who are ignorant of anatomy, and do not take the trouble to attend to it, are in no doubt at all.

His autopsy reports are preceded by a history and examination of the patients, and are often followed by a commentary on the salient features with references to Italian, Dutch and English authorities. He gives many accounts of symptoms, signs and *post mortem* appearances of pneumonia, empyema, pleurisy and pulmonary tuberculosis. He describes a Stokes-Adams attack and the morbid anatomy of mitral stenosis. The confirmation of the diagnosis was by way of the *post mortem* room, but however little could be done to stay the inevitable death of the patient, history and clinical observation and their correlation with morbid anatomy were paving the way for rational diagnosis. Morgagni died in 1791 at the age of ninety, and thirty years before Auenbrugger had published his little book of ninety pages on percussion of the thorax. Forgotten for nearly fifty years, some happy chance brought it to the notice of Corvisart, under whom Laennec studied in Paris, and the former translated it into French in 1808.

In 1819 Laennec published his "Treatise on Mediate Auscultation". In this edition he pursued the analytical method, giving the different signs elicited by percussion and auscultation, with the corresponding lesions. In the second edition, in 1826, the process is turned about and the method is synthetic, each disease being described in detail in respect of diagnosis, pathology and treatment. This edition is incomparably the more important. It is not a work on physical diagnosis, it is the most important treatise on disease of the heart and lungs ever written. What physician, before or since (he died at the age of forty-five), has packed into a short career a personal record of close clinical observation correlated, it is said, with over 26,000 autopsies—and that when he was suffering from pulmonary tuberculosis.

I wish that this survey could include something of the contribution of English medicine to clinical science up to the time of Laennec; and particularly of Thomas Sydenham, "the English Hippocrates", who fought on the side of the Roundheads, and thereby rendered himself ineligible for election to the Royal College of Physicians. Observation and experience led him to study what he called "the natural history of disease". Hippocrates, Morgagni and Laennec (who knew intimately the writings of his predecessors) bestride two thousand two hundred years—Hippocrates the master of prognostic, Morgagni seeking after rational diagnosis, and Laennec

building on these foundations what is the structure of modern clinical medicine. Sydenham advanced beyond the stage where morbid anatomy, diagnosis and prognosis could satisfy his ardour. We learn from him that, however much we may be interested in some single phenomenon of a particular disease, as practitioners of the healing art we must have as the objects of our endeavours in the individual case the sufferer and what we may do for him. To do this as best we may calls for every available piece of information which the patient can give us, and which we can draw from him by observation and rational deduction. Lest it may be thought that "what we may do for him" means something active, let me quote Thomas Sydenham:

> For I do not think it below me or my art to acknowledge with respect to the cure of fevers and other distempers, that when no manifest indication is pointed out to me what should be done, I have consulted my patient's safety and my own reputation most effectually by doing nothing at all.

The Present Position in Relation to the Past.

Perhaps this is a point at which one might take stock and consider whether there is a moral to be drawn from the development of the study of physical signs. I believe that it justifies the view that they are a means to an end, and not an end in themselves; that they should not be isolated from their context, the patient; and that, for instance, cavernous breathing does not necessarily mean a cavity, nor bronchial breathing, consolidation. Still less do cavernous breathing and bronchial breathing mean tuberculosis because they happen to be heard in a certain area. I think that most of the text-books on physical diagnosis are ill-balanced and even pernicious. I am certainly in revolt against their suggestions that such and such a sign is found in early tuberculosis, and their forgetting or neglecting to add that when it is present, other signs, more important, are also to be found; that more often than not it is not there at all; and that if it is there alone, it probably has no diagnostic significance. If we base our deductions from percussion and auscultation on the theory of sound, then in order that they may be of scientific value, we should draw the correct inference from them in the majority of cases. But too often the result is like the tale told by an idiot—full of sound and fury signifying nothing. I believe that Laennec may have had an idea of this, and so in the end he forsook analysis and turned to synthesis.

What superstructure have we built since Laennec? Ætiology and a more complete pathology—yes. But what of physical signs and our evaluation of them? Of heart disease Lewis said:

> I have tried to strip my subject of intricacies and redundancies, of unnecessary technical terms, named signs, and the old trite phrases, for these begin to stifle medicine.

Now if this be true of cardiology, how much more is it true of diseases of the lungs?

Radiological Examination as Revealing Physical Signs in Diseases of the Chest.

I believe that if we are to make progress along these lines we must include the findings of radiological examination of the chest among our physical signs. I like to regard it as the last step in physical inspection. But it reveals more than a sign, and in this particular domain, short of actual examination at operation, it is the nearest way in which we may approach to what Moynihan long ago called "living pathology". It should have revolutionized our conception and our teaching of physical signs, yet it seems scarcely to have made an impression. At Brompton Hospital it was my work to have the care for many months of over 200 artificial pneumothorax patients, and for a more variable period of between 400 and 500 such patients. I examined these patients with the screen at every visit before their refills, made an X ray examination of their chests every three months, and at shorter intervals if the occasion arose. Many of them had an X ray history extending over years, and so it became possible to gauge in some measure the time taken for disease to spread and for the formation of cavities and to watch the genesis, progress, or arrest of disease in the opposite lung. With the knowledge that I had gained I found a new interest in studying radiograms of the in-patients when later I had to see all the patients admitted to the public wards, and again at sanatoria. I have seen my conclusions verified or refuted at operation, by effluxion of time and by study at autopsy. Locke, writing of Sydenham, said:

> Nicely to observe the history of diseases in all their changes and circumstances is a work of time, accurateness, attention, and judgement.

I have gone but a little distance on the way, but my doubts (and they are many) and my beliefs on which I act are the result of my own observation, and that is, I think, as it should be.[1]

I have shown some films and have talked a little of the bedside signs which went with them. How does this affect my use of clinical observation? I know now that I cannot rest satisfied, although I try to find out by such means everything which the study of radiological appearances (including lipiodol injections) teaches me may be present. I hope that in some diseases at least I have indicated what this is. I wish to know, for example, whether in bronchiectasis the disease is unilateral or bilateral, confined to one lobe or not, and which one; and whether the dilatations are cylindrical or saccular. At one time it was enough to know at autopsy. Now I wish to know during the life of the patient, and I wish to follow the natural history of the disease. By combining history and bedside examination I try to determine these things. Nothing less will do as a diagnosis, because upon the knowledge of these points depends the treatment. When I have finished I know that the diagnosis is still uncertain, and I must have an X ray examination made.

In lung abscess I wish to know whether it is an abscess, single, localized and walled off, or not. In what lobe is it situated? Is it central or peripheral? And what is the position of the related bronchus? A few weeks ago the word went round among the students that a patient with a lung abscess had been admitted to the wards. A number of final year

[1] Some fifty lantern slides were shown at this stage, illustrative of various diseases affecting the lungs, mediastinum and pleural cavity.

students came to examine the patient. This commenced at once and in a few minutes, after the manner of the Arabs, they folded their stethoscopes and silently stole away. Others were warned, as they came along, that the findings were not typical—there were no physical signs. Twenty minutes' sitting by the bedside, listening to the patient's story, with the history of pain which had long ceased, the cough and sputum affected by posture, a look at the patient's clubbed fingers, and a lift of the lid of the sputum pot would have told them the diagnosis and would have given them a very good idea as to where the abscess was.

In pleurisy with effusion I wish to know firstly that there is fluid in the chest, then the size of the effusion, and what is the state of the underlying lung and of the other lung.

In spontaneous pneumothorax, is the condition of a ball valve type, or is the leak already closed? Is there fluid, and what is its nature? What is the state of the underlying lung and of the other lung?

In pulmonary tuberculosis is the disease unilateral or bilateral? Are there cavities present? How old is the disease, and, if it is bilateral, which is the more recent infection, and therefore probably the more active? Finally what is the state of the mediastinum and of the pleural membranes and cavities?

My examination finished, I fill in on diagrams of the chest the results of my observations. Then, having reviewed these signs in relation to the history, I make a comment as to what I think they suggest in terms of pathological findings revealed by radiology, and what is the probable diagnosis. I have, I confess, still an uneasy feeling that I may be far short of the actual state of affairs, either in the direction of understatement or of over-statement. Nevertheless, I should not like to leave out the physical examination of the chest by bedside methods and jump to the X ray picture. When I have done this, I have often wished that I knew what was to be found on percussion and auscultation. I cannot subscribe to the view that films should be interpreted without reference to the history and bedside signs, any more than that one should make an examination of the patient with the stethoscope without first being cognizant of the history. Sometimes it is history, sometimes physical examination which may predominate in giving us the information we desire; but if anything is going to offer us a means of increasing our knowledge of the natural history of disease in the chest, then I am convinced that it will be radiology. The more knowledge we can gain of natural history, the better shall we be equipped to judge the course which a disease is likely to follow, and to decide whether to institute treatment or to do nothing; and, what is at least of equal importance, the better shall we be able to gauge the result of whatever measures we introduce.

Pulmonary Tuberculosis.

Pulmonary tuberculosis bears out these statements better than other diseases of the chest, though it applies with force to such conditions as lung abscess and bronchiectasis. About pulmonary tuberculosis, I should like to say a little more, and particularly in regard to its early diagnosis. Satisfactory X ray pictures of the chest have existed for not more than ten years. Before that time it was customary to suspect that a young person might be suffering from this complaint on the evidence of certain symptoms. The next step was to evaluate the signs—hæmoptysis, temperature records and the result of the bedside examination. From the last, a whole literature of diagnostic signs arose, some of which is still extant. It is open to doubt whether we are able to diagnose early pulmonary tuberculosis with any certainty. This is not always our fault, because the disease process antedates those symptoms which are likely to bring the patient to the doctor.

Hæmoptysis is a dramatic event, and when not preceded by symptoms is commonly regarded as occurring very early in the disease. In the last century Niemeyer taught that it caused pulmonary tuberculosis. Time and again have I seen an X ray picture of disease which must have been in progress long before this first hæmorrhage, and often at a second attempt it is possible to obtain a history of earlier slight symptoms. Paradoxically, I believe I have seen recurrent hæmoptysis occur as the sole evidence of disease which is otherwise arrested. It is unsafe to diagnose pulmonary tuberculosis, even in the face of hæmoptysis, when tubercle bacilli cannot be found in the sputum and the X ray examination reveals no abnormality. I have seen so many hæmoptyses for which no cause could be found, even after the patients had been followed for two years.

I have little faith in the value of temperature records, though I read that a careful record for three weeks is a valuable diagnostic sign. I have demonstrated the spread of the disease in afebrile patients. I have worked in sanatoria where it is the custom to take temperature records four times daily by rectum and by mouth for ten minutes, and have observed the same process. I have seen a patient's temperature settle with a week's rest in bed, and have had cause to regret later that I did not produce an artificial pneumothorax. I have for three years followed patients whose temperature records by these accepted standards should have indicated that tuberculosis was present, and have found them in good health with no evidence of disease at the end of that time. How then can it be a diagnostic sign if the sputum is free from tubercle bacilli and there is no evidence of disease in the X ray films?

You may tell me of cases in which hæmoptysis preceded the development of signs revealed by X rays. I too have seen this. You may tell me of cases in which a patient has had a positive sputum and no X ray evidence. I know of them, too, but I think I could safely count them on the fingers of two hands. In this connexion, does it not seem a little strange that we should be content with the examination of a stained smear to decide so vital a matter?

I feel that there is something in what Dr. John Brown said of Sydenham:

> Human life was to him a sacred, a divine, as well as a curious thing, and he seems to have possessed through life, in rare acuteness, that sense of the value of what was at stake, of the perilous material he had to work in.

Perhaps it is for some such reason that one would rather risk missing pulmonary tuberculosis occasionally than diagnose it when it does not exist. This is not to say that I am not often doubtful, and that I would not in doubtful cases keep the patient under observation.

I wished to prove for my own comfort that there was some basis for the beliefs I have expressed. To this end I analysed 2,260 consecutive out-patient histories at Brompton Hospital. The notes were three years old. Three groups of patients were selected from this number—those with well established disease, those whose disease might be considered early, and those whose disease was doubtful. There were 545 cases: 388 in the first group, 64 in the second, and 93 in the third. The early and doubtful groups Dr. Roles and I analysed further. In the early cases all the patients had positive radiograms (except two with positive sputum, both of whom were well three years later); and some of them had a positive sputum also. Their average age was twenty-five years, and at a conservative estimate the disease in most of them had been in progress many weeks when they were first seen. In the doubtful cases we wrote to the patients when they were not still under observation. We analysed their in-patient records when they had been indoors for observation, and whenever possible we had them up for reexamination, including further X ray examination. The difficulties of tracing the ninety-three patients whose condition was doubtful were great, but in the three years which had elapsed we could discover no cases in this series which would lead us to doubt that positive sputum and X ray findings, although not infallible, were far in advance of other signs of pulmonary tuberculosis.

The Training of Students in the Diagnosis of Chest Diseases.

May I conclude with some thoughts on the training of students in the diagnosis of chest diseases? I do not suppose there is any branch of medicine in which students go out more badly trained. In England I remember Dr. Hope Gosse's telling me that he would not give students chest cases for their final examination, because they were so hopelessly astray in them. In the fourth year it is necessary to teach students physical signs as such, and perhaps rightly to be rather dogmatic about those signs which rest on a tolerably secure basis. I have tried to bring out the fact that observation and true deduction are the processes which have been responsible for whatever progress has been made in the study of these diseases. If there is a further process then it is a synthesis from the facts which have been deduced. To mix a metaphor, I am mindful of the fact that the medical course is not designed for the scratch player; but surely to have as our object the teaching to students the Socratic method of observing and of thinking for themselves is not so treating it. The converse is aptly illustrated by Epictetus:

> As if sheep after they have been feeding, should present their shepherds with the very grass itself which they had cropped and swallowed, to show how much they had eaten, instead of concocting it into wool and milk.

A student, not long before his final examination, will speak of a "patch of bronchial breathing", in the secret hope that the meaning of this sign will be discovered by his interrogator. If taxed, he will say that it should mean consolidation. When asked to what the consolidation is due, perhaps he ventures that from its position it is possibly due to tuberculosis. The next step is to ask him to reconsider the history, the general appearance of the patient and various other points which may be evident, and which he should logically have considered. Probably it will transpire that there is nothing wrong with the patient's lungs. Again, he will talk in a mystical fashion of "rise of pitch", and I sometimes think that Dogberry's remark that "they that touch pitch will be defiled" is susceptible of more than one meaning. Laennec described what he saw and heard. I believe that we should try to do the same; that we should be taught to realize that the best text-book is clinical experience, and that the object of all this history taking and examination has but one aim: a diagnosis which holds every available item of relevant information to enable us to prognosticate satisfactorily and to treat patients; and this cannot be put down in one or two words, as the text-books necessarily do. The final question that the student must be taught to answer is: "What is wrong with this patient?" not "What do you find in this patient's chest?" To his reply, "bronchiectasis", or whatever else it may be, the next question is: "Why?" The answer to this shows whether he has learned to reason soundly from his premises.

Knowledge comes, but wisdom lingers, and I have laid myself open to the charge of presumption, and worse—that I have spoken when others present are better qualified by experience and seniority. Perhaps I have not brought out clearly what I would. I believe I have some special qualification which may excuse me in part, and it is that, like Jacob, who served for another purpose, I served seven years for my degree. Perhaps, in my eagerness for those who are following in my footsteps, I am urging them on to reach that happy state which usually comes to us for a short time when we are house physicians, and which is best expressed in the words of Job to his comforters, when he said: "Lo, mine eye hath seen all this, mine ear hath heard, and understood it. What ye know, the same do I know also. I am not inferior to you."

If I have invested the shadow show of radiology with more reality than seems justified, I must add that there are many films I can neither understand nor explain. I take some comfort from Eddington's remarks in his lectures on "The Nature of the Physical World", when he says that "proof is an idol before whom the pure mathematician tortures himself. In physics we are generally content to sacrifice before the lesser shrine of plausibility". If, at times, my enthusiasm for a physical sign should run too high, there comes to mind the rebuke administered long ago: "An evil and adulterous generation seeketh after a sign; and there shall no sign be given to it."

Summary.

1. Physical signs are defined and considered in relation to clinical science.

2. An endeavour is made to trace the development of clinical science from Hippocrates to Laennec.

3. The present status of physical signs is examined.

4. Radiology is considered as presenting physical signs in diseases of the chest.

5. Pulmonary tuberculosis is considered as an example.

6. The teaching of physical signs to students is discussed.

Acknowledgements.

I am indebted to the physicians and surgeons of the Brompton Hospital for permission to use most of the films demonstrated, and to Dr. Roles for his help in analysing the cases mentioned.

Legends to Illustrations.

FIGURE I.—In the first interspace, on the left side, beneath and below the clavicle is a small shadow which represents an early stage in the spread of pulmonary tuberculosis. This was detected on screening the patient. The date on which it appeared could be accurately determined. It corresponded with what the patient thought was a mild influenzal attack. The duration was only thirty-six hours, and, when seen a week later, he was quite well and symptom-free. There were no physical signs other than of the pneumothorax on the right side. The natural history of these shadows has been followed by Wingfield in serial films.

FIGURES II, III and IV are from a series representative of a two-year history of pulmonary tuberculosis.

Figure II shows infiltration in the right upper zone. There were no physical signs. The patient was examined by X rays because of hæmoptysis. She had been examined four months before because of indefinite ill-health, but no film of the chest was taken.

Figure III is taken six months later. She had just returned from a sanatorium. She had gained weight and felt well. She had had no fever during her stay in the sanatorium. Careful physical examination by two observers disclosed no physical signs in the left lung. Figure III shows that there is a soft walled cavity just below the anterior end of the first rib, and there is also infiltration in the space below this. The method of spread is in all probability as in Figure I.

Figure IV, taken twelve months later, when the patient has been at rest for that period. The disease in the right lung is contracting upwards. In the left lung no definite cavity can now be seen. The character of the disease has changed. Fibrosis predominates, and the latest film taken shows that Nature is at work attempting her own thoracoplasty.

FIGURE V.—A film of a girl of seventeen who complained of lassitude for six weeks and pain in the right side. Examination rightly disclosed the pleural effusion, but failed to demonstrate the infiltration of the underlying lung. This patient was afebrile when seen, and continued so in hospital.

FIGURE VI.—The examination of the patient accurately disclosed that the infection was bilateral, that there were cavities in the upper zones of both lungs, and that there was a right pleural effusion. The film further shows that the septum between the upper and middle lobes is being drawn upwards. Note the sharp lower border to the dense shadow in the right upper zone. This represents the septum which normally lies at a lower level and is more transverse. It is permissible to deduce that the disease probably commenced in the right upper zone where the fibrosis is most marked; that it spread thence to the left upper zone; and that there is more recent infiltration in the middle and lower zones of the right lung, and pleural effusion. Though not of importance in this case, it is often wise to endeavour to estimate these points. They have an important bearing upon the decision as to whether a pneumothorax should be induced when the disease is bilateral, and more rarely as to whether a thoracoplasty is indicated. Bilateral disease *per se* is not necessarily a contraindication.

FIGURE VII.—Mediastinal hernia. Note the convex border in the left lung extending downwards from the anterior end of the second rib. It represents the edge of the right lung which has passed across the anterior mediastinum. It is best seen on screening in the phase of expiration. The mediastinum itself is not greatly displaced. In this case the pressure in the right pneumothorax was positive, but it may occur at times when the pressure is negative, and militate against successful control in pneumothorax treatment. In this case an endeavour was being made to cause collapse of the cavity which is present in the right upper lobe. The importance of the mediastinum as a splint is demonstrated by this film.

FIGURE VIII.—Collapsed upper lobe due to obstruction of the related bronchus. The physical signs are often very typical. It will be seen that the trachea is deviated towards the collapsed lobe and probably accounts for many of the signs. The chest movement is diminished over this area. The percussion note has a tympanitic quality. The breath sounds suggest a cavity, but no adventitious sounds can be brought out. These latter signs are probably to be explained by the proximity of the trachea to the collapsed lobe.

THE EARLY DIAGNOSIS AND TREATMENT OF INTRACRANIAL INJURIES OF THE NEW-BORN.[1]

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IT is still very surprising how many times such conditions as intracranial injuries of the new-born are incorrectly diagnosed as congenital heart disease or asphyxia or some other vague condition. We must learn to realize that these injuries occur much more frequently than is usually suspected, irrespective of the type of labour, and that they occur not only in the difficult, prolonged and instrumental deliveries, but also in many other labours.

The very severe cases of intracranial injuries are usually recognized. It is the more common and milder cases that are often undiagnosed and so untreated, and although the child may live, it will be subject to the risk in later life of the mental and physical impairment which is so characteristic of this unfortunate type of patient. I do not think that anyone now questions that a considerable proportion of all defective and crippled children owe their disability to these injuries inflicted during delivery.

Frequency of Injuries.

Actually it is only by careful and complete necropsies performed on all dead new-born infants, by the special Benekes technique, that will give the accurate information as to the frequency of intracranial injuries. Yet, although the great majority of cases do not reach the autopsy room, the presence of injuries can be diagnosed. I shall give you some figures which will give you an idea of the frequency of these injuries at *post mortem* examination. In one series of 107 consecutive natal and neonatal deaths in one hospital in England, to which a large number of emergency midwifery cases were admitted, I found from a study of necropsies performed as a routine, that in 46% of patients intracranial injuries had been present. This figure is consistent with those of other observers in other parts of the world. In view of the huge mortality associated with the condition (actually there are as many intranatal and early post-natal deaths from these injuries as from all other maternal and fœtal diseases together), and the frightful morbidity attendant on it, we should always be prepared to suspect cerebral birth trauma in the slightly abnormal new-born, and thus pursue the right line of treatment as early as possible. It is only by bringing this subject to the notice of the general practitioner, and impressing on him its frequency and importance, that an improvement in the early diagnosis and treatment of the condition can be expected. If he watches for these cases he will recognize them. Some obstetricians think that to achieve a live birth is all that is required; but the obstetrician must learn that, before he can congratulate himself on having a living mother and

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on April 15, 1936.

a living child after a difficult delivery, he must remember that as the result of this difficult delivery the living child may unfortunately develop into a very defective child. The attending obstetrician must thoroughly appreciate his responsibility for the early recognition of these injuries in the new-born, especially since therapeutic measures, to be effective in these cases, must be employed early.

Causes of Injuries.

Rather than enter into a lengthy discussion of the ætiology of these intracranial injuries in the new-born, I think that we can take it for granted that a mechanical factor is the direct cause of the injury, which is due to alteration in shape and compression of the fœtal head. This is accompanied by stretching of the supporting apparatus, particularly the *tentorium cerebelli*. This mechanical effect of alteration in shape and compression is greatest in cases in which there is excessive moulding, or in which the change in shape of the fœtal skull is accomplished too quickly, either by the natural forces of labour or by an operative delivery. I think I should mention that a few observers believe that cerebral hæmorrhage in the new-born is simply another manifestation of the hæmorrhagic diathesis; but, except in a very few cases in which this may be an important factor, I am certainly not in accord with this explanation, although it seems to be more reasonable than another suggested cause of hæmorrhage in the new-born, namely avitaminosis.

I shall not go fully into the subject of prevention of these injuries, although anyone interested may read a previous published article.[1] But as an aid to the early diagnosis of these injuries, one must understand the type of case in which they are most likely to be present. Naturally, diagnosis depends on symptoms and signs presented by the infant, but it is important to realize that the history of the labour may lead us to suspect these birth injuries. Obviously, any condition which may produce sudden and excessive stress on the fœtal skull in its passage through the maternal pelvis is likely to cause such injuries. Contracted pelves or oversize of the fœtus, rigid soft parts, difficult forcep deliveries, precipitate labours and breech extractions are factors which are likely to predispose to birth injuries. The too early application of forceps, before the head has had time to undergo a certain amount of moulding, is likely to result in injuries, although one must certainly not carry the dictum of masterly inactivity too far, since an unduly prolonged labour probably exerts a far more injurious influence on the child's brain than the skilled application of forceps. If we have ignored the danger of a prolonged perineal stage (which is certainly responsible for quite a fair percentage of our fœtal deaths) and then, as a last resort, forceps are used, we must not blame the forceps for the intracranial injury should one result. But serious injuries can be expected as the result of injudicious application of the forceps, such as in high forceps deliveries, most cases of mid-forceps deliveries, and also in cases in which the forceps have been applied in wrong diameters, especially in the antero-posterior diameter, that is over the forehead and occiput, or in an unrotated occipito-posterior diameter. So it is not so much in the cases in which we have applied forceps that we expect to find these injuries, but in the cases in which we have applied them wrongly. We have to learn not to throw indiscriminate blame upon the use of the forceps, but rather to blame the way in which the forceps were applied, or the fact that their use might have been rendered unnecessary by more careful ante-natal treatment.

In these cases we have to learn to suspect the increased possibility of intracranial injury, and so to be on our guard should the infant show any signs or symptoms suggestive of such injury. Again, in breech deliveries we have to be on the look-out for these complications, as the percentage of intracranial injuries occurring in these deliveries is usually very high. If there has been undue haste in delivery of the head—prompted by the usually unnecessary fear of fœtal asphyxia—then intracranial injuries in the infant may be expected.

Similarly, in a vertex presentation, if the delivery has taken place too rapidly, more especially in a precipitate labour or after the injudicious use of pituitrin, one must expect to find these injuries. Lastly, intracranial injuries should be suspected in premature infants. It must be stressed that premature infants are notoriously prone to develop intracranial injuries, no doubt owing to the abnormal fragility of the vessel walls and of the *dura mater*.

So in the attempt at diagnosis of intracranial injuries of the new-born, accurate details of the labour must be ascertained. Make an examination for any external evidence of excessive pressure on the cranial contents, such as extensive caput formation, excessive moulding, or forceps marks, especially in cases in which the forceps have been applied in the wrong diameters.

Diagnosis.

Signs and Symptoms.

There is not the slightest doubt that many infants who survive have intracranial injuries. The greater number of them will have no more than small hæmorrhages and small tears of the *tentorium cerebelli*, or probably more commonly œdema of adjacent parts. But others may survive who have extensive intracranial injuries; and I have seen infants who have survived for as long as eight days in whom *post mortem* examination revealed an old hæmorrhage as big as a fist and extensive damage of the dural septa. But as a general rule, if the hæmorrhage is extensive, the child is still-born.

In those who survive, the clinical signs and symptoms are neither uniform nor characteristic. They are usually present at or soon after delivery; but occasionally the symptoms do not appear for three or four days, because bleeding continues slowly for the first few days and the accumulation of blood eventually becomes great enough to produce signs or symptoms.

The infant is usually born in a condition of asphyxia, generally of the pallid type, and is often difficult to resuscitate. If respiration is established, it may be noticed in the next few days that the respirations are

slow and irregular, more rarely they are rapid and shallow, and there are often attacks of cyanosis. Often the child is noticed to have focal or general convulsions, neck rigidity, perhaps unequal pupils, and strabismus or nystagmus; it may be either extremely lethargic or very restless and emit a continuous whining cry. A most important point is that the sucking reflex is often impaired or absent.

Condition of Anterior Fontanelle.—The condition of the anterior fontanelle is very often misleading. It must be strongly emphasized that we cannot rely on the bulging of the fontanelle for diagnosis. Although in the greater number of cases of hæmorrhage the fontanelle is tense and bulging, occasionally it may even be depressed; yet autopsy may, when the fontanelle is depressed, reveal definite hæmorrhage, usually of the infratentorial type. Actually in such cases we usually find that the fontanelle has a more or less characteristic board-like feel. Attempts have been made to distinguish supratentorial bleeding from that of infratentorial origin by symptomatology; for the seat of the hæmorrhage is important, since a small blood effusion beneath the tentorium is of graver consequence, owing to close proximity to the vital medullary centres, than one on its surface, however large it may be. A supratentorial hæmorrhage characteristically occurs in the restless, irritable type of infant with bulging fontanelle, and with cyanosis that appears late and that is not especially pronounced since the respiratory centre is not affected until towards the end. In the infratentorial type, however, the infant is quiet, apathetic and early shows signs of cyanosis owing to close proximity to the medulla; and neck rigidity is soon present as the result of the irritating presence of blood in the upper part of the spinal canal.

Actually, as shown by autopsies, in the great majority of cases of intracranial injury hæmorrhages both of the infratentorial and supratentorial types occur, so that it is not usually possible to decide just where the hæmorrhage is likely to be. As regards the diagnosis in general, the most important thing that I can say is that, as long as the obstetrician thoroughly appreciates his responsibility for the early recognition of the injuries, and carefully observes the new-born infant immediately after birth and during the first few days, he will probably not miss the infant's suffering from these injuries, and so will be able to carry on with treatment immediately.

Lumbar Puncture.

To confirm our suspicions we have a relatively easy method, namely lumbar puncture. Lumbar puncture not only determines the diagnosis, but is of definite therapeutic value; for as the result of the relief by lumbar puncture, of the increased intracranial pressure, the danger of later impairments can be lessened and even avoided. But therapeutic measures to be effective in these cases must be employed early, at a time when the intracranial hæmorrhage can be drained in fluid form. Practically all authorities agree that the presence of blood-stained cerebrospinal fluid obtained by lumbar puncture is unmistakeable evidence of the presence of the intracranial hæmorrhage, except that a bloody fluid clearing after the first few drops must be attributed to the trauma of the lumbar puncture itself. As a rule, when port wine-looking fluid is obtained, there is no mistaking it for the red blood resulting from the puncture of a vein with the needle. We must appreciate the point that a colourless fluid does not rule out an intracranial hæmorrhage, although it is exceptional to meet with such cases.

In cases showing bloody spinal fluid, puncture repeated at intervals, sometimes three times a day, depending on the amount of hæmorrhage present, should be performed; and it may be stated that decompression by lumbar puncture can be carried out with safety, regardless of the degree of hæmorrhage. Actually, the amount of decompression which may be carried out is often surprising. In two cases in which I performed cisternal punctures twice daily for five days I removed as much as fifteen cubic centimetres of bloody fluid in some of the taps; yet a follow-up of these two cases after six months showed both infants to be apparently normal. In many cases, after puncture the results are prompt and very gratifying. Often the convulsions disappear completely, respirations tend to become normal, and other signs diminish as a result of the puncture and the relief of pressure; no untoward effects are shown. It is essential in the performance of the puncture, naturally without an anæsthetic, that the spine should be well flexed in the horizontal position, and that a very fine lumbar puncture needle should be used. If these measures are taken, the number of unsuccessful taps are few. In the odd case with a dry tap in a suspected birth injury, one may resort to puncture of the *cisterna magna*, which is really an easy method of obtaining fluid; but the technique is acquired only by care and understanding.

As a further means of diagnosis, the injection into the cerebral vessels of fluid impermeable to X rays has been attempted; but this procedure has not yet been sufficiently developed to be of service in the new-born, except for purposes of diagnosis in a dead infant when autopsy is refused.

Treatment.

It must once again be emphasized that it is only by early recognition and prompt treatment of intracranial injuries that the patient can be given the best possible chance of recovery and of normality in the future.

The value of lumbar puncture has already been stressed, but we must carry out other treatment before resorting to it. From the first, it must be emphasized that small hæmorrhages may soon prove fatal if unnecessarily aggravated by vigorous manipulation during resuscitation. So the new-born infant must not be swung vigorously by the feet or squeezed or manhandled by numerous brutal text-book methods in the first ten minutes after birth.

The failure of the new-born infant to breathe is usually due to depression of the medullary centres, and its failure to respond to resuscitation must certainly not be treated by these antiquated and unscientific methods, for they have in many cases

been the cause of an extension and diffusion of an already existing small localized hæmorrhage.

Practically the only treatment that should be practised in the resuscitation of the new-born is to remove any mucus from the throat by means of a mucus catheter, and to encourage attempts at respiration by gentle traction on the tongue with tongue forceps at the rate of about twenty pulls per minute, together with the injection of a cardio-respiratory stimulant such as "Coramine" or "Icorol". Should the infant attempt to breathe, carbon dioxide may be of some value.

If the infant does not readily respond to this treatment, then there is a likelihood of intracranial hæmorrhage being a factor contributing to the delayed establishment of respiration. The obstetrician must realize that practically all asphyxiated new-born infants which do not respond to this gentle treatment within a minute or two must be considered as having potential brain injuries and small hæmorrhages may soon prove fatal if vigorous manipulations are resorted to.

While the tongue manipulations are being carried out, the infant should be left in a warm bath or in warm blankets.

If the infant with injuries responds to this treatment, it should then be placed in a warm cot, preferably in a darkened room. It should not be put to the breast until all apparent danger is over, but should be bottle fed. Since the sucking reflex is frequently impaired or even absent in these cases, it may be necessary to feed the child for a few days by means of a pipette, or even through a catheter used as a stomach tube.

Attacks of cyanosis may be treated by the use of oxygen and carbon dioxide.

Besides lumbar puncture, the intracranial pressure may be lowered by rectal injections of about two ounces of 10% saline solution, given every four to eight hours until symptoms subside. This is an excellent prophylactic procedure in cases in which intracranial damage is suspected, even though no symptoms have yet appeared. I have noticed, however, that if the injections are given over too long a period, the infants become so dehydrated that they are likely to die from this cause.

When slowly continuing hæmorrhage is suspected, or occurs in association with the lumbar puncture, it is probably of value to inject about ten cubic centimetres of maternal blood into the infant's buttock, with the hope of diminishing the bleeding time.

Operative Measures.

Different operative measures have been suggested to relieve the condition of intracranial injury, but operative results have been far from satisfactory. Obviously this is because most operations today are performed at a time when the condition of the child is practically hopeless, and also because most cases are not suitable for the removal of large clots or the checking of the hæmorrhage, for the hæmorrhages are often multiple and it is often impossible to determine their exact location.

As a conclusion, I might suggest that the attention of the medical practitioner and of the midwife has only to be aroused to bring to light many cases of slighter intracranial damage. Since it is the obstetrician who sees these cases early, and not the pædiatrician or the neurologist, he must realize his great responsibility, for it is only by early diagnosis and prompt treatment that good results can be hoped for; and such good results must imply a decrease in the neonatal death rate and in the risk of the child's developing any of the many unfortunate handicaps which are known to be caused by these injuries.

Reference.

(1) R. H. Nattrass: "Prevention of Intracranial Injuries of the Newborn", *The British Medical Journal*, October 27, 1934, page 766.

MASSIVE PULMONARY EMBOLISM.

By J. Burton Cleland, M.D.,
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In three thousand autopsies at the Adelaide Hospital, forty-one examples of massive pulmonary embolism were met with. Ophüls,(1) in three thousand *post mortem* examinations in California, met with only ten examples; Rosenthal,(2) in Chicago, in one thousand consecutive autopsies, noted two. It would thus seem that massive pulmonary embolism is considerably commoner in Australian than in American experience. It can hardly be suggested that more care is taken to ascertain the presence of this condition in Australia than in America. I think it can therefore be inferred that massive pulmonary embolism is the cause of death much more frequently in Australia than in America. If this is so, there must be an underlying cause, and were it possible to ascertain and to remove this, a definite saving of life might be achieved. The present communication will serve to draw attention to the subject. In our experience in Adelaide, cases of this condition tend sometimes to occur close together over a period of time, and again no examples may be met with perhaps for many months.

Sex and Age.

Twenty cases were in males and twenty-one in females. As approximately about three *post mortem* examinations are done in men to two in women, this suggests a definite greater liability to the condition in women. The youngest patients were aged thirteen and a half and nineteen years; there were three in the twenties, seven in the thirties, three in the forties, twelve in the fifties, thirteen in the sixties, and one in the seventies. This distribution more or less follows the relative number of cases in which *post mortem* examinations were done at these various age periods, with the exception that there are an undue number under twenty, and a high proportion in the thirties.

Associated Conditions.

The following summary indicates the more important associated conditions: Post-operative, 14; after trauma, 3; pre-operative, 1; in pulmonary

tuberculosis, 3; in pneumonia, 2; in typhoid fever, 1; in Flexner dysentery, 1; in other infective processes, 3; after miscarriage, 1; in carcinoma, 1; from renal or suprarenal tumours, 2; from clots in the large veins of the leg, 4; in miscellaneous conditions, 5.

In Post-Operative Conditions (14).—These were equally divided between men and women, and the patients comprise the following: A woman of sixty-three following operation for epithelioma of the vulva and failure of the wound to heal; a woman of fifty after partial hysterectomy for prolapse; a woman of thirty-eight following removal of the Fallopian tube, and a woman of thirty-four eleven days after operation for pyosalpinx; a woman of thirty-four, operated on for chronic appendicitis with abscess formation; a woman of fifty-six operated on for acute appendicitis; a man of sixty-five with an infected suprapubic cystotomy wound after operation on the prostate; a man of sixty-nine three days after prostatectomy; a man of fifty-eight following cystotomy for urethral stricture; a man of sixty-eight, operated on for carcinoma of the bladder; a man of fifty-six, operated on seven days previously for inguinal hernia; a man of sixty-five with gastro-enterostomy for pyloric ulcer; a woman of sixty-four on the second day after partial gastrectomy with gastro-jejunostomy for carcinoma; a man of fifty-two who met with an accident, some months later the left leg was amputated for gangrene, and he died twelve hours after the amputation.

After Trauma (3).—A man of fifty-one had thrombosis of the veins of the leg following a bullet wound. A man of forty-five died eleven days after fracture of the femur. A boy, aged thirteen and a half, received a contusion of the stomach, followed by perforation and abscess formation; during anæsthesia a clot was set free and he died from massive pulmonary embolism; the thymus weighed 19 grammes.

Pre-Operative Case (1).—A man of sixty-eight, with an enlarged prostate, died suddenly just before operation from massive pulmonary embolism, the source of which was not detected.

In Pulmonary Tuberculosis (3).—Two of the patients were women, aged twenty-five and thirty, and the other a male aged fifty-eight. In both the women the clots were derived from iliac veins. The pulmonary tuberculosis was extensive in each case.

In Pneumonia (2).—Both patients were males, aged fifty and fifty-nine, the younger of whom had had bronchopneumonia with thrombosis of the right femoral vein; the other patient was convalescing from lobar pneumonia.

In Typhoid Fever (1).—The patient was a young man, aged nineteen.

In Flexner Dysentery (1).—The patient was a woman of sixty-three, who also had fatty infiltration of the heart and atheroma of the coronary vessels.

In Other Infective Processes (3).—A woman of seventy had cellulitis of the calf following ulcers and thrombosis of the posterior saphenous vein. A woman of twenty-one had acute appendicitis, and the appendix had a gangrenous tip. And a woman of thirty-nine had cellulitis of the arm, due to *Staphylococcus aureus* and thrombosis of the axillary vessels.

After Miscarriage (1).—A woman, aged forty-five, had thrombosis of the right internal iliac vein, old mitral stenosis, a ball thrombus in the left auricle, small infarcts in the spleen and probably an infarct in the brain.

In Carcinoma (1).—The only case was that of a male, aged thirty, who was not operated on and who had two separate carcinomatous masses in the sigmoid colon.

In Renal or Suprarenal Tumours (2).—A male of sixty-one had a Grawitz tumour of the kidney that had grown into the renal vein, from which a thrombus was later dislodged. A girl of twenty-three had a malignant growth of the right suprarenal gland with thrombosis extending into the *vena cava*.

Clots in the Large Veins of the Leg (4).—These comprise one man aged sixty-two and three women aged fifty-four, fifty-nine and sixty-six, who had thromboses in the popliteal and femoral veins in three cases, and varicose veins in the leg in the fourth.

Miscellaneous Cases (5).—A male of thirty-eight had a right hydronephrosis and a clot in the right internal iliac vein. A man of sixty-seven, with cerebral hæmorrhage, also had massive pulmonary embolism and infarcts in the lung and kidney. A woman of fifty-four had rupture of an atheromatous ulcer of the aorta, with hæmorrhage into the mediastinum. A woman of forty-two suffered from an obscure condition; she had suppression of urine and congestion of the brain. And a woman of fifty-seven had some abdominal symptoms and a very large pear-shaped gall-stone.

Period after Operation or Severe Trauma.

In fourteen cases information is available as to the date of operation or of severe trauma which had preceded the fatal pulmonary embolism. The days that had elapsed were as follows: 2, 3, 5, 5, 6, 7, 8, 8, 10, 11, 11, 13, 15, 16. It thus appears that massive pulmonary embolism may occur as soon as the second or third day after a severe abdominal operation, though the majority of cases centre round a period of about ten days. There were no cases in lying-in women in the series under review.

The Period of the Year and Intervals between Cases.

As an impression had developed that cases of pulmonary embolism tended to occur in groups, the exact dates at which each occurred were analysed, but the distribution of cases seems to be entirely due to chance. The following cases occurred:

1920: 2—September 9, November 12.
1921: 8—February 2, March 23, April 2, July 6, July 23, October 8, October 9, November 26.
1922: 3—February 21, July 31, November 21.
1923: 0.
1924: 1—December 23.
1925: 3—January 26, July 16, November 5.
1926: 3—January 24, July 8, October 20.
1927: 1—October 19.
1928: 2—May 10, September 20.
1929: 2—February 16, September 21.

1930: 2—November 18, December 6.
1931: 4—May 9, June 30, November 14, December 15.
1932: 7—February 4, February 21, February 21, March 27, April 22, September 29, October 1.
1933: 2—April 26, May 1.
1934: 1—July 29.
[1935: 4—May 3, August 10, October 24, November 10.][1]
[1936, to May: 2—January 22, April 14.]

To see whether any clear view could be obtained from the interval between cases, which might show any tendency to grouping, I have estimated roughly, in days, these intervals. The figures are not exact, being only approximate when I have converted months into days. The intervals are as follows: 49, 75, 30, 10, 60, 17, 75, 1, 45, 90, 150, 120, 690, 34, 180, 90, 75, 180, 105, 365, 210, 120, 150, 210, 420, 18, 150, 49, 135, 30, 58, 17, 0, 34, 26, 150, 2, 210, 5, 450, [270, 90, 75, 17, 75, 90].

To see whether there was a special tendency for these cases to occur in certain months of the year, I have arranged them accordingly, as follows:

January 24, 26=2. [January 22.]
February 4, 16, 21, 21, 21, 26=6.
March 23, 27=2.
April 2, 22, 26=3. [April 14.]
May 1, 9, 10=3. [May 3.]
June 30=1.
July 6, 8, 16, 23, 29, 31=6.
August 0=0. [August 10.]
September 20, 21, 22, 29=4.
October 1, 8, 9, 19, 20=5. [October 24.]
November 5, 11, 12, 14, 18, 21=6. [November 10.]
December 6, 15, 23=3.

Here again the distribution of cases seems entirely haphazard.

Summary.

1. Massive pulmonary embolism occurred in approximately 1·3% of three thousand autopsies at Adelaide. This is much more frequent than in American records. No explanation as yet presents itself for this frequency.

2. Women seem more liable to this complication. The condition may occur at any age from childhood upwards.

3. One-third of the cases were post-operative. The condition may also occur in pulmonary tuberculosis, after pneumonia, in typhoid fever, after other infective processes, and from the extension of renal and suprarenal tumours.

4. After operation or severe trauma, massive pulmonary embolism occurred from the second to the sixteenth day.

5. There is no evidence that any period of the year is more liable than another to pulmonary embolism, or that cases occur in groups sufficiently closely for some common factor to be operating to increase the liability.

References.

(1) William Ophüls: "A Statistical Survey of 3,000 Autopsies", Stanford University Publications, "Medical Sciences", Volume I, Number 3, 1933.

(2) S. R. Rosenthal: "Thrombosis and Embolism: Analysis of 1,000 Autopsies", *Journal of Laboratory and Clinical Medicine*, Volume XVI, November, 1930, pages 107-118.

Addendum.

Since the third thousand *post mortem* examinations were completed, 300 further autopsies have been carried out and massive pulmonary embolism has been met with in six. Three of these followed operations as follows. A woman of fifty-seven had cholecystectomy performed ten days previously for gall-stones. A man of fifty-two died nine days after a similar operation for gall-stones; *ante mortem* clots were found in the left external iliac and femoral veins and branches of the popliteal. A man of forty-four died six days after operation for strangulated inguinal hernia. A woman of sixty died nine days after a Pott's fracture of the right leg, with a thrombus still present in the right posterior tibial vein. A woman of fifty-two had a large Grawitz tumour of the right kidney which had grown into the renal vein. A man of thirty with a right-sided pneumonia and empyema, died with a massive pulmonary embolism, and had thromboses of the right external iliac and hypogastric veins at autopsy.

[1] In this and the following lists the data between brackets refer to cases mentioned in the *addendum*.

Reports of Cases.

CONCURRENT CHRONIC INTUSSUSCEPTION AND RETROGRADE INTUSSUSCEPTION.

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R.W., AGED seven months, was admitted to Saint Vincent's Hospital, Lismore, at 6 p.m., on May 18, 1936. His mother gave the following history. About 11 a.m. on April 5, 1936, while sitting on the floor playing, he suddenly screamed with pain, went white and sweated. He was a breast-fed baby and up to the time of this attack had been perfectly well. After the attack passed he went to sleep. During the day and the following night he had several screaming attacks, which were all similar to the first. The next morning he passed some dark blood and slime *per rectum* and was at once taken to a doctor at about 8 a.m. A bismuth mixture was perscribed, on the supposition that his condition was due to enteritis.

During the following week he had several slight attacks of screaming. He vomited occasionally. The bowels were opened every day, but no blood was passed.

Eleven days after the first onset, the same sort of attack came on late in the afternoon. Screaming attacks occurred frequently (every five or ten minutes, his mother said) all night and next day. He vomited bile occasionally and refused food. He passed small motions of blood and slime all day with the attacks. He was given a mixture containing phenazone and he got quite well and took his food again.

A week later a third attack began, but was not severe. It lasted from midday till about 7 p.m. and he then recovered. About this time he was given powders containing one-third of a grain of calomel, on the supposition that his digestive disorder was due to teething.

Five days later he had a fourth attack, when only one screaming spasm occurred, and his fifth and sixth attacks were separated by two-day intervals from those preceding them. None of these attacks was severe, but they were quite characteristic. Following these he had slight attacks two or three times a week until two days before his admission to hospital. During this time, since the commencement of the illness, he had lost 1·3 kilograms (three pounds) in weight.

On May 16, two days prior to his admission, at about 5 p.m. a very bad attack began. This time he did not pass so much blood and his mother noticed a lump in the left side of his abdomen. Slight attacks continued all that night and the following day, and he was passing small amounts of blood and slime. He vomited everything taken by mouth. On May 17, at about 6 p.m., he became very ill and had screaming attacks every two or three minutes. He was now passing more blood and slime than at the first onset of his illness. Castor oil was given on May 17 and also on May 18 without result. An enema of olive oil was given at about midday on May 18, and also produced no result. The attacks continued all day on May 18.

The patient was admitted to hospital at about 6 p.m. on May 18 with a note stating that he was suffering from intussusception and that this was palpable *per rectum*. When I saw him he was very pale, lethargic, and appeared to be semi-conscious. The eyes and cheeks were sunken and the hands and feet were very cold. The patient's temperature was 36·5° C. (97·8° F.), the pulse rate was 176 per minute, and the respiration rate 36. The abdomen was slightly distended and a very firm, tense mass was palpable in the left iliac fossa and extending up almost to the left costal margin. *Per rectum* the head of an intussusception could be felt about 3·75 centimetres (one and a half inches) from the anus. No blood was seen, but mucus was present in fair amount. There were no other abnormal findings.

It was decided to operate as soon as possible, and while preparations were being carried on an attempt was made to secure some reduction of the intussusception by means of hydrostatic pressure. This produced no result, which is not surprising in view of the subsequent findings. Dr. J. L. Roberts, of Lismore, saw the child with a view to the administration of an anæsthetic, and in consideration of its very bad condition he suggested that an attempt should be made to perform the operation under local anæsthesia.

Injection was accordingly commenced about 7 p.m. with 0·5% "Procaine" and 1 in 200,000 adrenaline in normal saline solution. Intercostal nerve block of the seventh to twelfth dorsal nerves was effected on both sides, combined with subcutaneous and subaponeurotic infiltration of the line of incision. A mid-line incision 10·0 centimetres (four inches) in length was made with its centre at the umbilicus. The intussusception, with its advancing head in the rectum, was felt and was found to be irreducible. On further investigation a tense mass in the left flank was with some difficulty withdrawn from the abdomen and proved to be a retrograde intussusception in the descending colon. It was about 15·0 centimetres (six inches) long and about 5·0 centimetres (two inches) thick. Reduction was difficult, but after compressing it with the hands for some minutes it slowly began to unfold. Reduction was completed and the head of the retrograde intussusception, though very much swollen, œdematous and discoloured to a purple tint, was considered to be viable. The primary intussusception was then very readily reduced by simple finger pressure as far as the splenic flexure of the colon. There a little difficulty was met, but was soon overcome and reduction was continued till the final swelling was gently squeezed out. The intussusception was of the ileo-caecal variety, and, apart from some œdema, the ileum and caecum appeared quite normal after reduction. The wound was closed by three through-and-through silkworm gut sutures, catgut mattress sutures for the peritoneum and aponeurosis and dermal suture for the skin. Apart from crying at the preliminary intradermal injections, the baby took the operation remarkably well and was quite easily amused throughout. Its condition appeared to be much better than prior to operation, probably owing to the stimulating effect of the adrenaline in the local anæsthetic solution. It showed signs of nausea (but did not vomit) two or three times towards the end of the operation, which was finished at about 8 p.m.

At 10 p.m. that evening the child's condition caused anxiety. The pulse was still very rapid and weak, and any movement, as for nursing purposes, caused a falling off in the general condition. It was very pale and restless. The bowels had moved and a small stool of tarry, altered blood had been passed. It was taking teaspoonfuls of water and glucose frequently, and the extremities were now warm.

On May 19 its condition was much improved. The bowels had moved well on several occasions and the child was taking small feeds of breast milk every two hours. The temperature had risen to 38·3° C. (101° F.). On the evening of May 20 the temperature rose to 38·9° C. (102° F.) and respiratory infection became evident with cough and snuffles. The chest was clear. The child was irritable and apparently was troubled by teething pains. During the next couple of days the respiratory infection subsided. The bowels moved normally, but the motions were very green, with sometimes mucus and a little undigested curd. Powders of calomel, 0·015 gramme (one-quarter of a grain), twice a day gradually cleared up this condition. The child recovered well and left hospital on the sixteenth day.

Comment.

The most striking feature of this case is the history of typical acute attacks of intussusception occurring so frequently over such a long period as six weeks in an infant seven months old. The mother's description of the attacks was very clear and intelligent, and there is no doubt of the number of attacks characteristic of acute intussusception that the child suffered. The history was typical of acute intussusception from the beginning, except that, as sometimes happens, blood was not passed *per rectum* until about twenty hours after the first onset. In this matter of the passage of blood this child appears to differ from the majority of those children reported as suffering from chronic intussusception. Still,[1] describing the typical case of chronic intussusception, writes: "There has been no blood in the stool, or at most there has been only a streak once or twice, such as might be seen on the stool of any constipated child." Owen,[2] in reporting a case of four weeks' duration in a child of six years, states that no blood was passed at any time. The same writer mentions that no blood was passed by Schlink's patient of seven years of age with a two months' history, reported in 1922. Clubbe[3] records eight cases of chronic intussusception, the two longest of which were of thirteen days' duration, but does not mention the passage of blood. For these reasons I regard this case as more acute than most cases of chronic intussusception.

Another possibility is that recurrent attacks of acute intussusception occurred and that the invagination was reduced between the attacks. This I find difficult to believe, on account of the number and frequency of the attacks, and also by reason of the fact that following operation, at which no attempt was made to prevent further attacks, the child's condition cleared up completely and it has remained quite well.

The second remarkable feature of the case was the presence of a retrograde intussusception of the descending colon. This condition is evidently very rare. Lockhart-Mummery[4] writes: "Retrograde intussusceptions do occur, but only during death or as the result of asphyxia; they are not met with in practice." Ochsner[5] writes that ascending or retrograde intussusception has been reported, but is extremely rare. Lake and Marshall[6] apparently regard retrograde intussusception as occurring only in the dying. Clubbe[7] records two cases of retrograde intussusception, only one of which is comparable with the present case, the other presenting three or four small retrograde intussusceptions in the small intestine. Considering that at the area of the retrograde intussusception there are five layers of bowel wall between the outer peritoneal coat and the central lumen, and in view of the tenseness of the tumour here, I do not think that the retrograde intussusception can have been present for more than forty-eight hours. It was at this time that the really bad final attack commenced and the mother noticed the presence of the lump in the patient's abdomen.

A fact which I think deserves emphasis is the extreme value of regional and infiltration anæsthesia in a case such as this. The child's condition prior to operation was so serious that it was not considered likely that it would survive general anæsthesia, at any rate any general anæsthesia that was available. Yet not only did the injections produce no deleterious effect, but the patient appeared to improve considerably during operation in spite of the extensive handling that was necessary and the time required for the operation. There was not the smallest sign of muscular resistance, pain or shock at any stage of the operation.

In regard to the diagnosis of intussusception, one suggestion might be made which, I think, may be useful. In those cases in which the history is strongly suggestive and in which no blood has been passed *per rectum* after some hours, the use of a rectal injection of water or

saline solution may reveal the presence of blood. This finding not only confirms the diagnosis, but is of very great help in convincing the parents that an apparently healthy baby is urgently in need of operation. In February last I was called to see a girl of eight months. The history was of an acute screaming attack at 10 a.m. that morning. The mother telephoned me about 3 p.m. and there was apparently no urgency in the matter. I saw the child at 5 p.m. and elicited the fact that no blood had been passed by the rectum. A small rectal injection of water resulted in the passage of a quantity of blood. Operation was undertaken at 6 p.m., when the intussusception had reached the splenic flexure of the colon. Once the presence of blood in the bowel had been established the mother was only too anxious for operation to be proceeded with.

Summary.

1. A case of chronic intussusception in a child aged seven months is described, the condition being of six weeks' duration.

2. A retrograde intussusception was found at operation in addition to the primary intussusception.

3. The value of regional and infiltration anæsthesia in this case is pointed out.

4. A suggestion is made regarding the earlier diagnosis of acute intussusception in cases in which blood is not passed after some hours.

Acknowledgements.

I am indebted to Dr. J. L. Roberts, of Lismore, for his assistance and the loan of literature, and to Dr. A. J. Opie, of Lismore, for one of the articles referred to.

References.

(1) G. F. Still: *Clinical Journal*, January 25, 1922, page 37, quoted in "The Medical Annual", 1923, page 232.
(2) A. W. Owen: "Remarks on the Diagnosis and Treatment of Chronic Intussusception", *The British Medical Journal*, May 24, 1924, page 904.
(3) C. P. B. Clubbe: "Diagnosis and Treatment of Intussusception", Second Edition, 1921, page 28.
(4) P. Lockhart-Mummery: "Diseases of the Rectum and Colon", 1923, page 294.
(5) A. Ochsner: "Nelson's Loose Leaf Surgery", Volume V, page 268.
(6) N. C. Lake and C. J. Marshall: "Surgical Anatomy and Physiology", page 562.
(7) C. P. B. Clubbe: *Loco citato*, page 85.

Reviews.

ALLERGY AND LYMPHADENOMA.

In his thesis "Allergy in Relation to Lymphadenoma", G. P. Chandler attempts to throw some light on the nature of Hodgkin's disease by searching for some allergic phenomenon associated with it.[1]

In a brief and very interesting historical review he mentions most of the theories which have been evolved, and concludes that, although Hodgkin's disease may ultimately prove to be an infective process, no satisfactory evidence has been advanced so far in favour of any one organism as a cause. Nor does the evidence favour the explanation that it is an atypical form of tuberculosis or a newgrowth. He next reviews the existing knowledge on hypersensitiveness and devotes a separate chapter to bacterial allergy. He concludes that allergy can be demonstrated in a very large number of bacterial diseases and in time will probably be shown to occur to some extent in all.

For his research he has attempted to isolate some allergenic principle from the glands in Hodgkin's disease and to show by intradermal tests that the patient is responsive to this. Several different methods were used in obtaining these gland extracts, but trial of these in seventeen cases gave almost uniformly negative results. Apparently reactions were looked for at twenty-four, forty-eight and seventy-two hours, but presumably the result of the test was also observed for an hour after injection, as there would be the possibility of some immediate response. Attempts to demonstrate immune bodies in the sera of seven patients when extracts of lymphadenomatous glands were used as an antigen likewise gave negative results.

Chandler concludes that, although the histological appearances of affected glands and certain clinical features suggest that Hodgkin's disease is an infective process, his researches show none of the allergic characteristics associated with infections. If they have any significance they may possibly lend slight support to the neoplastic theory.

This research appears to have been well planned and carefully carried out, and although his findings were negative, the author's treatise will form a useful addition to our knowledge of this disease.

[1] "Allergy in Relation to Lymphadenoma", by G. P. Chandler, M.D., M.R.C.P.; 1934. London: John Bale, Sons and Danielsson. Demy 8vo, pp. 111. Price: 10s. 6d. net.

PHYSICAL SIGNS IN CLINICAL SURGERY.

In eight years Hamilton Bailey's "Physical Signs in Clinical Surgery" has proceeded to the fifth edition, an indication of popularity justly deserved.[1] As in the preceding editions, the author has kept down the size of the book by keeping the description of methods short and by relying on the really excellent photographs to illustrate the methods which he favours and teaches.

It would be difficult to criticize this work. Perhaps it might be stated that in the sections dealing with the examination of the acutely inflamed abdomen many signs are described which are seldom seen. But to offset this minor complaint is the excellence of the sections dealing with the upper and lower limbs and with hernia.

Possession and use of this book are a necessity for a clinical dresser; and in spite of the profusion of photographs—no fewer than three hundred and forty-one—the price has been kept well within the means of any student, a fact not always borne in mind.

[1] "Demonstrations of Physical Signs in Clinical Surgery", by H. Bailey, F.R.C.S.; Fifth Edition; 1935. Bristol: John Wright and Sons, Limited. Royal 8vo, pp. 299, with illustrations. Price: 21s. net.

OPERATIVE SURGERY.

It is pleasing to record the arrival of the first volume of "The Operations of Surgery", now edited by Rowlands and Turner, but still modelled on the original and well-proven Jacobson's book of the same title which appeared close on fifty years ago and which has been taken as the standard of surgery at Guy's Hospital.[2]

The main objects of the original are adhered to, namely, to provide real assistance to young surgeons and to those wishing to study for higher examinations, and to provide a convenient source of reference to the busy surgeon. In addition, credit and fair criticism are given to new methods that seem worthy of trial.

The volume under review deals with the upper extremity, the head and neck, the thorax, the lower extremity and the vertebral column, and coincident with the advance of surgery many of the older methods are replaced or remodelled according to modern technique. It is interesting to note how experience in the Great War has altered what used to be standard procedures, especially as regards

[2] "The Operations of Surgery", by R. P. Rowlands, M.S., F.R.C.S., and P. Turner, B.Sc., M.S., F.R.C.S.; Eighth Edition; Volume I; 1936. London: J. and A. Churchill Limited. Royal 8vo, pp. 1055, with 435 illustrations, of which 38 are in colour. Price: 36s. net.

amputations, nerve suture and traumatic lesions of the skull and brain; in fact a first class discussion on the last mentioned is included.

When certain procedures are mentioned only in brief, the full references to current literature are given, a feature which considerably enhances the value of the work.

There are certain parts, however, which would bear some amplification or even inclusion in the text, such as: (i) methods of skin preparation other than those of iodine and picric acid, (ii) the use of diathermy in dealing with cerebral tumours, (iii) Cushing's clips for the control of hæmorrhage from the cerebral vessels, (iv) the adoption of the modern classification of cerebral tumours and their diagnosis, (v) Royle's operation for cervical sympathectomy, (vi) the detail of lobectomy, (vii) the technique of thoracoscopy.

Apart from these few omissions, the work is a mine of information on unusual procedures and retains its classical chapters on operations for hare-lip and the ligature of arteries. The details of transfusion and infusion are good, and the conservative surgery of the hand is excellent.

There are still a few of the older procedures which could well be discarded. An instance occurs in the chapter dealing with trigeminal neuralgia, where it is stated: "All mere neurotomies and nerve stretchings are absolutely futile. Radical lasting cures by peripheral neurectomy are practically unknown." Yet considerable space is given to these very procedures and the detail for section of the fifth nerve root is meagre.

The value of radium in the treatment of carcinoma of the tongue is stressed, but the various classical operations for this condition are wisely retained.

The question of pre-operative X ray treatment of carcinoma of the breast is dismissed very summarily in the words: "the advantage of this is not obvious, at any rate as a routine measure, and it is not recommended"; but radium treatment is given in detail.

One feature of inestimable value to the younger surgeon is the discussion of operative difficulties and mistakes; and in many cases the end results of various procedures are mentioned as a guide to what the surgeon in all fairness may lead his patient to expect.

The publishers have every reason to be proud of their share in the production of this volume, and the printing, illustrations and paper are first class; also, although the text occupies over one thousand pages, the book is so bound that it will remain open at any page—a small point, but one which makes for the comfort of the reader.

Regarded from all points of view, it is a book which no young surgeon can afford to be without.

ALLANTOIN AND THE COMFREY.

SINCE ancient times medicinal virtues have been ascribed to the comfrey (*Symphytum officinale*, Linné). Macalister has published a brochure on its clinical uses, with an investigation of its chemistry.[1] It appears that allantoin is the active principle. Allantoin is present both in animals and plants, is found in the allantoic fluid, and has been identified in the urine of healthy persons. Macalister relates the striking effects of allantoin on plant growth and inflorescence. In human subjects ulcers show remarkable benefit, indicating the great value of allantoin as a cell proliferant. Allantoin cleans up sloughing surfaces in burns and ulcers generally, but has no definitely antiseptic properties. A good many cases are on record in which cancerous or sarcomatous growths appear to have been benefited by comfrey, but neither comfrey nor allantoin appears capable of producing a somatic cell from a malignant one, or a carcinomatous or sarcomatous cell from a somatic one, and no explanation is available for the occasional cures following their employment. It is known that the maggots of some flies, when placed on wounds, expedite their healing. It has been found that allantoin given off by the maggots is responsible, at least in part, for the curative effects. Recent observations suggest that allantoin may be beneficial by stimulating phagocytosis, and remarkable guinea-pig experiments are quoted. Pneumonia was accordingly treated by the administration of allantoin with very encouraging results. In normal individuals administration of allantoin was followed by a great increase in the polynuclear cells, with a return to normal in from sixteen to forty-eight hours. Macalister considers that allantoin may be useful in infective conditions which may be benefited by the production of a leucocytosis. The impression gained as to the cell-proliferating property of allantoin is that it has a hormone-like action. Small quantities initiate proliferations of considerable magnitude.

Macalister is enthusiastic as to the possibilities of allantoin. Further investigation will decide whether the optimism is justified. Dr. Titherley gives an account of the chemical constitution of allantoin, both its dextro-rotatory and lævo-rotatory forms. He discusses the theory of tautomerism. Allantoin is amphoteric. It is very sensitive to acidity and alkalinity as well as to high temperature. Allantoin not only occurs naturally, but may be synthesized.

[1] "Narrative of an Investigation Concerning an Ancient Medicinal Remedy and its Modern Utilities: The *Symphytum Officinale* and its Contained Allantoin", by C. J. Macalister, M.D., F.R.C.P., together with an Account of the Chemical Constitution of Allantoin, by A. W. Titherley, D.Sc., Ph.D.; 1936. London: John Bale, Sons and Danielsson, Limited. Crown 8vo, pp. 60. Price: 2s. 6d. net.

A MANUAL OF HYGIENE.

THE appearance of the sixth edition of "Elementary Hygiene for Nurses", by H. C. Rutherford Darling, is evidence of the popularity of this manual.[1] Dr. Darling, by presenting a new edition at three-yearly intervals, has kept his production up to date.

The volume deals in a very comprehensive manner with all the essential factors in hygiene. It contains eleven chapters. Five deal with the general hygiene principles of light, heat, air, sanitation of buildings, water, drainage, sewage, temporary hospitals *et cetera*. The remaining chapters deal with the prevention of infectious diseases, immunity, parasites and insects and personal hygiene.

The book is well produced and illustrations are adequate and well chosen, and it may be read with benefit by students and practitioners as well as nurses.

HEREDITY AND GENETICS.

IN "Adventures Before Birth" M. Jean Rostand, a distinguished French naturalist, undertakes an account of the processes by which a human life comes into being.[2] The germ cell, the sperm cell, their preparation for union, and the journey which enables them to meet are described. The ovum, the result of their union, is followed through various phases of growth; problems which are suggested by events in its evolution are indicated. Early development is given in considerable detail, the later stages necessarily being sketched in more broadly.

The book is interesting and stimulating, and the claim that it fills a need may well be substantiated. It has the advantage that it contains no propaganda or religious dogma and, written as it is with knowledge and imagination, it should help readers to a clearer understanding of the complexity of the problems of heredity and genetics. The translation is pleasantly done; here and there a phrase is reminiscent of other ways of speech, but the sense of the passage is not obscured.

[1] "Elementary Hygiene for Nurses: A Handbook for Nurses and Others", by H. C. Rutherford Darling, M.D., M.S., F.R.C.S., F.R.F.P.S.; Sixth Edition; 1935. London: J. and A. Churchill. Crown 8vo, pp. 358, with illustrations.

[2] "Adventures before Birth", by J. Rostand, translated by J. Needham; 1936. London: Victor Gollancz Limited. Crown 8vo, pp. 157, with illustrations. Price: 4s. 6d. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 5, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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A PRESIDENT'S ADDRESS.

THE address which was prepared by Dr. J. Tate Mason, President-Elect of the American Medical Association, for delivery at the annual meeting of the Association in May, is published in *The Journal of the American Medical Association* of May 16, 1936. It has a message to those who profess and follow medicine in all parts of the world. Taking as his subject "Modern Trends in Surgery", Dr. Mason has something to say about economic and social conditions before he refers to standards in surgery and its future development. Great changes are taking place in social, economic and political spheres, not only in the United States of America, but in all civilized countries. Dr. Mason thinks that these changes are bound to affect the medical profession as a whole, and we are all prepared to agree with him. He points out, however, that the problem of the medical practitioner is twofold. Not only has he to consider his own professional status, but he has as his responsibility the welfare of the general mass of citizens. Admittedly occasions will arise on which medical practitioners may be compelled by short-sighted people to maintain their rights and privileges as practitioners of the healing art. These are occasions to be regretted, for it may be taken as truth that if medical practitioners make the welfare of the general mass of citizens their first consideration, their status as practitioners will take care of itself. The average person, Dr. Mason states, has little conception of the problem of the welfare of the community and is influenced by all sorts of propagandists without any idea of the dangers that would follow from the adoption of various ill-considered schemes. This brings us to a principle that has previously been stated in these columns—that since medical practitioners have peculiar privileges of knowing and of studying certain needs of the community, those of their number who are temperamentally and otherwise suited for it should take an active part in the public life of the State. Moreover, those who do undertake this thankless duty should at least be immune from the envy and petty jealousy of their brother practitioners who remain less brightly illuminated.

In discussing the past, the present and the future of surgery, Dr. Mason shows that for fifty years surgery has concerned itself chiefly with the correction of the mechanical defects of the body. Diseased organs have been removed, obstructions have been corrected, tumours have been destroyed—procedures made possible by the practical application of knowledge concerning the anatomy, chemistry and physiological activity of the human body. The surgeon of today is no longer content with these procedures. His desire is to restore, if possible, the whole organism to its physiological harmony—in other words, reconstructive surgery. Technical skill will continue to be improved, and we must agree that there is no limit to mechanical ingenuity in the production of new instruments. "New instruments will not only improve the technical skill but as diagnostic aids will enable the surgeon to determine and correct disorders of function even before morbid processes develop." In all present-day medical writings more and more attention is being paid to the patient as a whole, to the restoration of what Dr. Mason calls the physiological

harmony of the individual. In the future the surgeon may be able to treat and to cure diseases now regarded as incurable; he will also in all probability find that diseases now treated by him may be treated more satisfactorily by non-surgical means. The surgeon thus appears to be situated somewhat similarly to the medical practitioner who enters public life—he lives more in the public eye than other members of the profession, mainly because his work is spectacular. He should be immune from the jealousy of those who have not taken the trouble to equip themselves to undertake surgical work. On the other hand, if reconstructive surgery is to be the surgery of the future, he will have to rely more and more on physiologists, biological chemists and pathologists, as well as on those who devote themselves to internal medicine. He may become more specialized than he is, but he will probably find that preventive measures will often make operation unnecessary. He will therefore not complain, since the aim of surgery is the prevention of surgery.

Dr. Mason's address might have been prepared by the president of a medical society in any part of the civilized world. Medicine, like literature, science and the arts, is international. In these days of suspicion, mistrust and jealousy among the nations emphasis should be laid on any common interests and objectives that they have. When many people, for example, are declaring that the League of Nations has been a failure, it is well to remember the great deal that has been done for the world by the Health Organization of the League and by the International Labour Office. Nations are composed of individuals, and the outlook of the individual must eventually determine the action of the nation. Underlying Dr. Mason's address is the idea of self-effacement in the service of science and humanity. This is what is needed most today.

Current Comment.

THE SEROLOGICAL DIAGNOSIS OF CANCER.

THE value of a reliable biochemical test for the diagnosis of malignant disease would be incalculable. It would not be confined merely to the diagnosis of individual cases, but would have a broader application, and would help no doubt in the illumination of some of the many obscure features of malignant disease. The Bendien test depends on the phenomenon, demonstrated by Bendien some five years ago, that protein vanadate is precipitated when a suitable acetic vanadate solution is added to blood serum. The reagent consists of 20 to 30 cubic centimetres of deci-normal sodium vanadate solution, to which sufficient deci-normal acetic acid has been added to make 200 cubic centimetres. Precipitation normally occurs when a reagent containing 28 cubic centimetres of vanadate solution per 200 cubic centimetres is used; it is greater, the greater the strength of the reagent. In some pathological conditions precipitation is increased and occurs when weaker reagents are used. Bendien believed that the precipitation in malignant disease was characteristic; but it has been found that there are many conditions in which similar reactions occur. Bendien found that, if serum was treated with ether, precipitation was increased, and if the serum was subjected to a temperature of 56° C. for thirty minutes, precipitation was diminished. Lowe therefore modified Bendien's test, comparing the increase in precipitation caused by ether treatment with the decrease in precipitation caused by the heat treatment of serum. This test and its results in 450 cases are discussed by R. Ewart Jones and D. Leyton Woodhouse.[1]

If the amount of precipitation from normal serum is designated *A*, that from heated serum *B*, and that from ether-treated serum *C*, the ratio may be expressed by the formula: $\frac{C-A}{A-B}$. In the paper by Jones and Woodhouse the formula is wrongly expressed thus: $\frac{C-A}{C-B}$; this causes annoyance and confusion to any person attempting to read the paper intelligently. Jones and Woodhouse observed what strength of reagent caused a precipitate of one milligramme of protein in untreated serum; they then compared the results obtained with reagents of the same strength mixed with treated serum. In malignant disease the ratio is supposed to be greater than one, whereas in the normal or in disease other than malignant disease, it is less than one. Though a comparatively weak reagent may cause a heavy precipitate in many non-malignant conditions, the ratio remains low. In diagnosis, therefore, no significance is attached to the amount of precipitation; reliance is placed only on the ratio. In the series discussed by Jones and Woodhouse the serological diagnosis of malignant disease agreed with the clinical diagnosis in 74·8% of 175 cases, and the serological diagnosis of the absence of malignancy agreed with the clinical diagnosis in 74·4% of 121 cases. A feature of interest is that the serological diagnosis was accurate (according to clinical findings) in 90% of cases when the

[1] *The Quarterly Journal of Medicine*, April, 1936.

patients were less than twenty years of age, and in 95% when the patients were aged twenty-one to thirty years. Lowe has drawn attention to the possible value of the test in prognosis and the evaluation of treatment. Successful treatment should cause a change in the reaction. Recurrence might be revealed by a serological test before it was obvious to clinical examination. Jones and Woodhouse followed up a few patients with the object of testing these ideas. They are unable to form any conclusions from such a small number of cases; but they believe that the results of their investigations are an encouragement to a further study of this aspect.

It is clear from a perusal of this paper that the vanadate test is by no means infallible; but the same may be said of many tests that are commonly employed and relied on in medical practice. Sole reliance should not be placed on any one test in the diagnosis of cancer or any other disease. If suitable serological tests are available, the medical practitioner should use them as he requires them, but only if he has a proper appreciation of their value. At present the value of serological tests for cancer is difficult to assess. At first glance a single test of paramount diagnostic importance in all the various types of malignant disease scarcely seems feasible. But all malignant diseases have at least the one common characteristic of malignancy; perhaps they have many others, possibly even a common origin. When these things are considered, the evolution of a common means of diagnosis becomes conceivable. A great deal of work on biological tests of malignant disease has been done in Germany; it is hoped that a special article dealing with this subject will be published shortly in this journal. If a test approaching the ideal is to be found, it will be by an intelligent correlation of clinical and biochemical knowledge.

THE POSSIBLE TRANSMISSION OF HÆMOLYTIC STREPTOCOCCI BY DUST.

ATTEMPTS to trace the source of puerperal infections have suggested very strongly that in the majority of cases the streptococci have been conveyed to the genital tract from the nose or throat of the mother herself or of someone in contact with her. In a minority of cases, when no such probable cause is found, the question of infection by dust arises. Elizabeth White[1] points out that in private houses at the time of a confinement "there must sometimes be persons with septic foci infected by these streptococci, such as children with otorrhœa or impetigo, or adults with infected wounds, who, although not in close contact with the mother during labour or in the puerperium, may have contaminated the dust, which in turn may originate a puerperal infection". In order to obtain further knowledge of the possibility of infection by dust, she has carried out a bacteriological investigation of the dust of those single bed wards at Queen Charlotte's Hospital which are reserved for patients with puerperal fever.

Tests on the air and the dust in the wards were carried out between the third and the sixth day after admission, as follows: Two horse blood-agar plates were exposed by the night nurse after she had settled the patient for the night. One was placed on the trolly about one and a half yards from the foot of the bed, and the other on the locker beside the bed. These were closed by the nurse when she woke the patient in the morning and were therefore exposed for about six hours. Two more plates were exposed in the same places by the nurse who made the first perineal toilet of the patient; they were left open while she was doing this and then closed. They were open for about twenty minutes to half an hour. It is stated that there would be more movement in the ward during this period than during the night. While the ward was being swept and polished two more plates were opened, one being placed on the trolly at the foot of the bed, as in the other cases, and the other on the floor. These were exposed for about twenty minutes. The six plates were incubated overnight in anaerobic conditions, because "this helps to keep down the saprophytic organisms and also shows up the hæmolytic colonies more clearly".

It was found that the hæmolytic streptococcus occurred in the dust of single bed wards where patients infected with this organism were nursed, and not to anything like the same extent in the wards of patients with sepsis due to other organisms. The twenty-seven patients, the dust of whose wards was tested, varied from a patient with mild localized infection of the genital tract to one with a local infection *plus* pelvic cellulitis, general peritonitis, puerperal scarlet fever and septicæmia. The degree of contamination of the dust did not appear to be related to the severity of the infection. In most cases the strain isolated from the dust was proved to be identical with that infecting the patient.

Two experiments were made to investigate whether the hæmolytic streptococcus would live in the dust of the room when the source of contamination was removed. These experiments seemed to show quite clearly that the streptococcus would live in the dust for several days. White quotes an experiment conducted by Leonard Colebrook, who found that when a broth culture of hæmolytic streptococci was sprayed into a dusty cupboard, the organisms remained there for ten weeks.

As a corollary of great practical importance, tests were made of the efficacy of the method employed for disinfecting the rooms after they were vacated by the patient. It was found that spraying with formalin (40% formaldehyde) destroyed the hæmolytic streptococci in rooms contaminated with that organism. When weaker disinfecting solutions were used, hæmolytic streptococci were found to persist on two of the five occasions on which the test was used.

[1] *The Lancet*, April 25, 1936.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

The Antagonism in the Development of Malignant Disease in Two Different Organs.

W. CRAMER (*The Journal of Pathology and Bacteriology*, July, 1936) has tried to determine whether the development of cancer in one organ influences in any way the development of cancer in a different organ. He has used two different strains of mice and has painted the animals with tar. The organs chosen for observation were the skin and the mamma. He finds, as previous observers have found, that tarring of the skin of mice of pure strains having a high percentage of mammary cancer yields a percentage incidence of skin cancer not higher than in mice which have a low incidence of spontaneous mammary cancer. There is evidence of an antagonism for carcinogenesis in the two sites, skin and mamma. The skin of animals which most readily develop mammary cancer is resistant to the carcinogenic action of tar. Conversely, individual mice in which the skin is most susceptible to the carcinogenic action of tar do not readily develop mammary cancer. This constitutes further evidence that the carcinogenic response of the skin to a carcinogenic agent, although in itself a strictly localized process, is nevertheless conditioned by systemic factors lying outside the area subjected to the agent. As a result of this relationship the simultaneous occurrence of skin cancer and mammary cancer is rare, and the incidence of skin cancer in tarred mice diminishes as the actual incidence of spontaneous mammary cancer increases. This may account for the statistical phenomenon observed in man, that different populations may show an approximately equal total incidence of cancer with a widely different organ incidence.

Generalized Lymphatic Carcinosis of the Lungs.

T. T. WU (*The Journal of Pathology and Bacteriology*, July, 1936) states that generalized cancerous permeation of the pulmonary lymphatics, known in the Continental literature as *lymphangitis carcinomatosa*, is a relatively rare condition. He reports five cases observed in the Department of Pathology and Bacteriology of the University of Leeds, and reviews 49 collected from available literature. Of the author's five cases, four were secondary to gastric carcinoma; this was the finding in three-quarters of the cases from the literature. In the author's fifth case the primary tumour was an adenocarcinoma of the right bronchus. Other sites for the primary tumour are the breast and prostate; rare sites are the uterus, sigmoid colon, gall-bladder, ovary and tongue. The chief subject for discussion is the route by which the cancer cells reach the pulmonary lymphatics. The generally accepted view is that retrograde spread follows involvement of the hilar lymphatic glands. In all the author's cases, with the possible exception of one in which there was no naked-eye evidence of cancer in the tracheo-bronchial lymph glands, the condition could be explained in this way. There are two other modes of producing lymphatic permeation of the lung. The cancer cells can reach the subpleural lymphatics of the visceral layer from the serous sac itself, with subsequent extension along the pulmonary lymphatics. The cancer cells can also, by way of the blood stream, reach the pulmonary arteries and become implanted beneath the pleura; from these secondary growths permeation of the lymphatics may follow. Although gastric cancer is very often associated with this unusual condition, the author gives reasons for his belief that gastric cancer is not biologically more prone than other forms of cancer to give rise to it. Obliterative changes of both thrombotic and endarteritic types were present in the pulmonary arteries in two of the author's cases. He presents evidence in favour of the view that the obliterative changes are to be attributed to the effects of cancer cell emboli and not to the mere presence of cancer cells in the perivascular lymphatics. Some of the extreme examples of this condition may simulate the clinical picture of Ayerza's disease. It has been pointed out that while the clinical features of Ayerza's disease are clear cut, the underlying pathological process may be quite different in individual cases. The author suggests that generalized cancerous permeation of the pulmonary lymphatics, especially when associated with obliterative changes in the pulmonary arterioles, is another condition capable of producing Ayerza's disease.

Hypertrophy of the Breasts.

H. WINKLER (*Monatsschrift für Geburtshülfe und Gynäkologie*, May, 1936) describes a case of diffuse hypertrophy of both breasts in a *nullipara* aged forty-eight years. The menstrual history had been normal and the excessive growth had been noted for two years. Coincident with the mammary growth the hair had fallen out and fat deposits had occurred in the thighs. There were no ocular symptoms, the *sella turcica* was normal in outline, and the basal metabolic rate was raised 2% above normal. Both breasts were amputated; the right measured 45 by 28 centimetres and weighed 4·2 kilograms, while the left was 39 by 28 centimetres and weighed 2·6 kilograms. Section of the breasts showed an increase of alveolar development, but no increase in secretion. The author found difficulty regarding the ætiology of the condition. Under hormonal influence hypertrophy at puberty may be noted, and it remains constant with no tendency to spontaneous recovery. More rarely it may first develop during pregnancy, disappearing after delivery, to return in future pregnancies.

Teratomata.

JOSEPH KRAFKA (*Archives of Pathology*, June, 1936) states that the theory of organizers, as elaborated by Spemann, gives a consistent and complete explanation of teratomata, covering the variation in morphology and the wide distribution that have so commonly been observed. The author discusses the polar body theory of Marchand and Ribbert, the blastomere theory of Bonnet, the cell rest theory and the theory of the parthenogenesis of germinal cells. He points out that these are all untenable. The author believes that interference with the normal effect of the organizer may result in the production of secondary axes in the primitive field and lead to the formation of a teratoma at any site. He explains the observed frequency of teratoma in man as a function of proximity of the potential fields to Hensen's node. He explains that the primitive knot or Hensen's node is generally accepted as the homologue of the dorsal lip of the blastopore, as seen in the salamander; and that differentiation of the primary vertebrate axis occurs from this point. Since no limit is placed on the degree of diffusion of the factor, the occasional occurrence of teratomata in the brain, umbilicus and placenta is explained. Mechanical interference prevents the occurrence of teratomata in the heart and liver, and the delay in the development of the limb buds until after the effective period of the organizer accounts for the non-occurrence of teratomata in the limbs. The association of chorio-carcinoma on a teratogenous base in both genital and extragenital sites is explained on the hypothesis that the amnion is potentially chorion.

Apophyseal Subluxation.

L. A. HADLEY (*The Journal of Bone and Joint Surgery*, April, 1936) describes two types of degeneration of intervertebral disk, that observed in early life, characterized by herniation of the *nucleus pulposus* into the adjacent intervertebral bodies or into the spinal canal, and another degeneration noticed in later life, characterized by fragmentation of the cartilaginous plate with fibrosis of the adjacent portions of the disk. Pain following these degenerations results from secondary alterations in the relationship between the adjacent structures. The degeneration of the disk decreases its thickness, thus bringing the vertebral bodies closer together and producing kyphosis or subluxation of the apophyseal articulation. The latter condition causes pain, which may be due to strain upon the fibrous tissue, decrease in size of the

intervertebral foramen in both diameters, fibrosis about the nerve roots with symptoms of radiculitis, or, in extreme cases, actual bony contact between the tip of the articular process and the pedicle above or the lamina below. The author also describes alterations between the contour of the edge of the vertebral body, the pedicle, lamina and transverse process. This apophyseal subluxation does not seem to take place in scoliosis, but is present in certain cases of lordosis.

Primary Abdominal Pregnancy.

W. NAGEL (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1935) describes a case of primary abdominal pregnancy in the spleen. The patient was a *multipara* who gave a history of irregular menstruation with slight uterine enlargement. Nothing abnormal was felt on abdominal palpation. A diagnosis of incomplete abortion was made and the uterus was curetted. Examination of the scrapings revealed normal premenstrual endometrium with no evidence of decidual cells or villi. During convalescence the patient complained of abdominal pain and collapsed, with signs of intraabdominal hæmorrhage. The abdomen was opened and found to be full of blood. The uterus and appendages were normal, and further exploration revealed placental tissue in the upper pole of the spleen and a 12-centimetre fœtus lying free in the abdominal cavity. The patient died on the table despite auto-transfusion. Examination of the spleen showed that the placenta was attached to it alone and had no connexions with surrounding structures. Full details of the microscopic examination of the spleen and placenta are given. The fœtus was fresh and showed no signs of maceration. The author claims that this is the first authentic case of primary abdominal pregnancy, and he is at a loss to explain how the ovum migrated to this area.

MORPHOLOGY.

Skulls from the Purari Plateau, New Guinea.

F. WOOD JONES (*Journal of Anatomy*, April, 1936) describes four skulls which came from the Purari Plateau, New Guinea. His summary of the main features of these skulls is as follows. The form of the calvarium in *norma verticalis* is ovoid, with a tendency to be somewhat angulated. There is no obvious cranial asymmetry in any skull, and the average cranial index is 75·6. In *norma facialis* there is some tendency towards keeling of the vault. The brow ridges are prominent and confluent in the mid-line. The orbits are square and oblique. The root of the nose is depressed, the bridge elevated. The narial aperture is wide, the average nasal index being 57·3, and the margins are guttered. The guttering of the narial margins is associated with a well-marked subnasal prognathism, the average alveolar index being 107; but the chin is fairly well developed and tends to be rounded. The occiput in *norma lateralis* is produced and not flattened, and the external occipital protuberance is extremely well developed. The forehead above the brow ridges is retreating. The teeth are large and well formed; the average length of the molar series is 28·5, the first molar being the largest. The "anterior root" of the zygoma is situated above the first or second upper molar, and the mental foramen is below the posterior edge of the second lower premolar. In general, the muscular impressions on the skull are extremely well marked, and the origin of the temporal is situated high up on the side of the skull. Racially, perhaps the most distinctive features of these skulls are the approach to mesaticephaly without flattening of the occiput; the width of the narial aperture and the marked guttering of its margin, combined with a prominent nasal bridge; the prominence, without great massiveness, of the molars; and the development of the chin in the presence of a high degree of subnasal prognathism. The average cranial capacity is 1,280 cubic centimetres. The author states that the people of the Purari Plateau have been estimated to number 200,000 or more. He adds that, as white contact is now established and is likely to prove disastrous to the natives, it is to be hoped that a thorough examination of the tribes by a trained and sympathetic anthropologist will soon be undertaken.

The Development of Tooth Germs in Vitro.

EXPERIMENTAL work has been carried out on the growth of isolated embryonic tissues *in vitro* with a view to studying the capacity of these tissues for self-differentiation. Although deprived of a vascular and nervous system and of connexions with neighbouring structures, many tissues develop in a comparatively normal way during cultivation. S. Glasstone (*Journal of Anatomy*, January, 1936) has carried out experiments to determine whether embryonic dental tissues will differentiate normally when isolated and cultivated *in vitro*. He used tooth germs of 18-day to 21-day rat embryos, and he gives details of the technique adopted. He found that whole or partial tooth germs showed remarkable powers of histological differentiation. The dentine papilla developed normally, forming odontoblasts which deposited normal tubular dentine. The presence of the internal enamel epithelium was essential for odontoblast formation. Dentine formation could take place in the absence of the enamel epithelium, provided that odontoblasts were present in the explant. One of the functions of the enamel organ is to determine the gross morphological structure of the tooth. The author found what he calls scar tissue in some of his cultures; this was similar to the early stages of the so-called scar tissue found in the teeth of guinea-pigs suffering from scurvy. He is investigating further the nature and significance of this abnormal dentine.

The Age of Epiphyseal Union.

M. J. S. PILLAI (*The Indian Journal of Medical Research*, April, 1936) has made a radiological study of the union of the epiphyses with the shafts of long bones. The age of union was observed in 100 Indians between the ages of ten and twenty-three years. The author found in his series that fusion occurred two to three years earlier than the ages published in text-books, "which appear to be the average for natives of temperate climates". He also noted that fusion occurred slightly earlier in girls than in boys. In conducting the investigation the author made radiographs of the elbow, wrist, phalanges, knee, ankle, foot and hip. He notes that his findings are similar to those of Hepworth, published in *The Indian Medical Gazette* in March, 1929.

A Myocardial Purkinje System in the Ventricles.

D. I. ABRAMSON AND S. MARGOLIN (*Journal of Anatomy*, January, 1936), using the injection method, have studied the conduction system in the heart of the sheep and the ox; in the heart of the dog and the pig they used histological methods only. They claim that their evidence shows that the anatomical concept of the ventricular conduction system, hitherto held, is incomplete. The Purkinje arborization of the ventricles, which is generally believed to be only subendocardial in location, ramifies throughout the outer ventricular walls and in the interventricular septum. This myocardial Purkinje network is directly continuous with and structurally identical with that of the endocardium. The authors call attention to the necessity for considering the myocardial Purkinje network in the evaluation of conduction phenomena in the ventricles.

A Double Human Pregnancy with a Single Corpus Luteum.

H. L. WIEMAN AND C. K. WEICHERT (*The Anatomical Record*, May, 1936) report an unusual double human pregnancy with a single *corpus luteum*. They obtained the specimen from the body of a women, twenty-five years of age, who had been wounded in the abdomen by a bullet two days previously. No second *corpus luteum* of pregnancy could be found. Two chorions were partially united by an intermingling of their villi. The implantation sites were widely separated. The authors discuss the origin of the embryos and conclude that it was biovular and not monovular.

Special Abstract.

TISSUE CULTURE.

MARTIN SILBERBERG[1] is responsible for the compilation of a lengthy paper which summarizes our present knowledge of tissue culture. In the fields of experimental biology, physiology and cytology the advances have been of the greatest importance, but Silberberg is mainly concerned with their significance in the domain of pathology. In the eighties of last century Roux demonstrated that the neural and intestinal canals of a chicken embryo continued to grow when placed in a weak solution of sodium chloride, and Loeb, in 1897, suggested suitable methods for the cultivation of isolated tissue particles. Later still, Harrison published important observations on the development of nerve fibres and upon the behaviour of embryonic tissues isolated in clotted lymph. All this work formed the basis for the methods of tissue culture elaborated during the past quarter of a century by Alexis Carrel, the chief of which has been the adoption of blood plasma as a medium. For this purpose plasma has not yet been supplanted. Its stability permits the continued growth and amœboid motion of explanted cells; without such stability in the supporting medium, the phenomenon of stereotropism, as the movement of the cell pseudopods has been called, ceases to exist. A knowledge of the general phenomena of explantation is of great importance in that by microscopic analysis of living matter the observer is at once free of the necessity for drawing conclusions from the observation of fixed and stained specimens of tissue. In explanted specimens the first sign of life is the movement, relatively rapid, of the explant's peripheral cells into the supporting plasma. This migration begins in a few hours and reaches its peak within twenty-four hours. The peripheral cells during the process multiply by mitosis, increasing greatly in size. After the third day the outgrowth is of considerable size, but in the central regions of the explant the processes of degeneration, necrosis and disintegration are now apparent. The growing cells by metabolic activity produce a steady liquefaction of the plasma, greater in proportion to the vigour of their growth. If the stereotropic phenomena are to continue, the explant must now be transferred to a fresh medium.

The cultivation of the mesodermal tissues, giving rise to adult connective tissues, is an easier task than that of epithelium. Epithelial growth is accompanied by excessive liquefaction in the nutrient plasma and frequent passages into fresh media must be made. Embryonic tissues in general grow extremely well, and the lower the animal from which the explant comes, the more vigorous the growth will be. Rabbit, chicken and guinea-pig plasma are the best media for the growth of explanted tissues; human plasma, for some reason yet undetermined, is not satisfactory. The rate of cell multiplication in explanted tissue affords no index of the age of the subject from which the tissue was originally taken. The specificity of cell growth remains constant, no matter how often transfers are made into fresh plasma. Three different types of tissue growth have been described: (i) the cytotypic growth of isolated cells, (ii) the histiotypic phenomena shown by membranous-like increase of epithelial explants, and (iii) the organotypic growth, to be observed in tissue containing epithelium with a supporting mesenchymal stroma, which results in the production of a rough organ-like arrangement in the explant. Up to the present time the tissues which have been successfully explanted are embryonic iridal epithelium, the lenticular layer of the skin in the chicken embryo, portions of the thyreoid gland (with an accompanying formation of colloid substance), pulmonary tissue, and liver substance capable of producing glycogen. In the matter of the last-named explants it has been observed that the hepatic tissue of the rat is cultivable more easily than that of the chicken and, further, that the epithelium of the bile ducts grows more vigorously than liver cells themselves. The point whether the bile duct epithelium is capable of conversion into hepatic cells, or *vice versa*, is not yet determined; the matter is of importance as regards possible regeneration after retrogressive metamorphoses, such as those which accompany yellow atrophy, cirrhosis and syphilis. Tissue explants of the pancreas, like those taken from the breast and salivary glands, tend to undergo a process of "dedifferentiation", to produce the general appearance of atypically growing cancer cells. This is noticeable in cells of the islands of Langerhans; epithelium from the pancreatic ducts grows well and in an orderly manner. A healthy explant of gastro-intestinal tissue from the chick embryo, by contractions in the muscularis, shows typical peristaltic movement. In the case of bronchial tissue the mucin cells quickly disappear or are converted into unspecialized epithelium, the cilia alone remaining at the end of nine days. Successful explantations have been made from specimens of the urinary bladder and gall-bladder of newly-born and adult animals. Organotypic growth of renal tissue has followed the cultivation of the embryonic metanephros of frogs and guinea-pigs. A membranous-like growth of the tubular elements occurs, but the convoluted tubules undergo rapid degeneration and only the collecting tubules survive. It is recorded that ammonia and urea have been produced in cultivated renal tissue. Survival, but no successful growth, follows explantation of portions of adrenal gland, and the secretion of epinephrine has been noted in these specimens. The continued growth has also been achieved of portions of testis, ovary, *corpus luteum*, human placenta and in the vitelline syncytium of the umbilical cord in the chicken embryo.

Mammalian nervous tissue, when explanted, behaves just as in the body so far as regenerative tendencies are concerned. The cerebro-spinal fluid is a good culture medium, and the cerebral cortex of the cat has been made to survive in it. The various parts of the pituitary gland have been explanted, and it has been shown experimentally that the *pars anterior* has no hormonal effect whatever; tissues from the posterior lobe and the *pars intermedia*, however, produce a rise of blood pressure and have a stimulating effect on the melanophore mechanism of the frog.

The blood, bone marrow and all hæmatopoietic and lymphopoietic organs have all been successfully cultivated, as have been specimens of thymus gland and of serous membranes. The growth is also reported of explants of fat tissue and of the cartilage, bone and periosteum of human embryos. Cartilage in fact has been kept alive during twenty-three passages and then reimplanted into an animal. Even more striking is the growth of explanted cardiac tissue. It grows well and shows differentiation of muscle fibres. It soon commences to show physiological pulsations. Different pieces of cardiac tissue taken from the same subject will, if explanted together, unite and pulsate synchronously. Further, the two specimens to exhibit these phenomena need not necessarily be taken from the same heart, but must be derived from the hearts of animals of the same species. The successful growth of skeletal muscle has also been accomplished.

Tissues affected by glanders, leprosy and tuberculosis have been cultivated outside the body, but negative results have followed attempts to grow material infected by spirochætes. Sections of nasal polypi, gliomata and of a hypernephroma have also been made to proliferate outside the body. As to cancerous material, much work has been done on the Ehrlich "mouse-breast" and upon growths of the Flexner-Jobling type, as well as upon malignant tissue from human beings. Repeated explantations of sarcomatous tissue taken from both the rat and from man have been accomplished.

With regard to the cultivation of immature organs, the development of the primitive heart into four chambers has been recorded; the normal toe-buds of embryonic chicks, when explanted, have been seen to undergo processes of growth similar to those in the body. The

[1] *Archives of Pathology*, May, 1936.

embryonic eye of the fowl is similarly capable of early differentiation into retinal rods and cones. Nerve cells, lens fibres and ciliary bodies also appear, but these processes soon cease.

A study of the finer morphology of the explanted cell shows that the movements of cytoplasm and nucleus are affected by pseudopodia. The cell membranes are invisible in reflected light or by dark-field illumination, but the use of the so-called "supravital" stains (janus green, janus black and others) has permitted observers to study the structure and functions of these and other cell components. By the use of carmine or pyrrhol blue, added either to the tissue before explantation or to the clotted plasma medium, it is possible to watch the processes of vital storage. These phenomena seem in general to follow the same principles *in vitro* as in the living body. Leucocytes alone take no part in storage, and fibrocytes and undifferentiated mesenchymal cells have but a slight capacity for it. A strange exception to the general rule is that the cells of the mesonephros and many types of epithelial cells show an ability to store vital dyes *in vitro*, a faculty which is not observed within the body itself.

Metabolism is always higher in explanted tissues than in normal tissues. This is due to the rich conditions under which they live, and is responsible for the fact that even under aerobic conditions they show great powers of glycolysis.

A study of the retrogressive metamorphosis occurring in explants reveals that the production of the pigment melanin is a function of certain mature and differentiated cells. The pigment is elaborated from fine colourless particles, and the mechanism of the conversion of these into melanin is as yet unexplained. But pigments have been produced by the disintegration of hæmoglobin and are to be seen, stored up in granular form, within the cytoplasm of the macrophages. In unstained and living specimens, too, hæmosiderin may be seen as a dark yellow or brown mass giving the characteristic microchemical reactions for iron. Various pigmented particles of exogenous origin have been added in the hope of elucidating such problems as that of pneumokoniosis. The added particles are shown to be digested by the macrophages, coal much more readily than other materials.

Colloid formation in explanted thyreoid tissue was first reported in 1913; this occurred if the cultures were not transferred for long periods. Thyreoid tissue from chicken embryos has now been kept alive for seven months, colloid formation having been noted after four months' growth, a possible demonstration that growth occurs in reverse proportion to the function of the tissues. With regard to the problem of fatty degeneration, the saturation of cultured cells with fat seems to run parallel with the increasing age and decreasing growth of those cells. But though lipoids may fill up the cytoplasm, mitosis may still proceed within the cell. Fat certainly appears under poor cultural conditions, but may disappear on the improvement of these. These facts may in time cause us to abandon the term "fatty degeneration".

Cultivated hepatic cells have been proved to be capable, but under favourable conditions only, of synthesizing glycogen. The production is directly proportional to the rate of growth, and is independent of the medium used. The glycogen exists in solution within the cell. Glycogen synthesis has also taken place in an explant of intestinal epithelium on the addition of dextrose to the culture medium. Yet the same production of glycogen may occur in an explant of the embryonic intestine of the guinea-pig without the addition of dextrose. Points for determination are not only whether glycogen synthesis occurs normally in cells other than those of the liver, but whether the production of glycogen in intestinal epithelium is the result of artificial conditions in the explant or a sign of the so-called glycogen degeneration.

Another unsettled question is that of keratinization. It may be indicative of a process of differentiation, but it certainly occurs in epithelial specimens in old culture, which, from their thickness, have suffered from lack of oxygen and of nourishment. From this aspect the change would appear to be degenerative in nature.

Calcification, known to have taken place in cultures of cartilage and osseous tissue, has also taken place to a much greater extent in a cartilage explant derived from a patient with *myositis ossificans*. In this instance there were also large precipitations into the connective tissues of the explant, so that after ten days large areas were completely calcified. It therefore appears that calcification depends upon the preexisting amount of calcium in the tissue.

Tissue culture has made possible a series of important observations upon the phenomena of growth, since isolated cells or groups of cells can be studied either in connexion with nerves or independently. Cultures grow better the oftener they are cut, and the features of growth *in vitro* are analogous to those characteristic of the healing of a bodily wound. Carrel, Fischer and many others consider that wounds elaborate certain hormones (variously called desmones, trephones and necrohormones), supposedly produced by the regeneration, necrosis and necrobiosis of cell material, and possessing a stimulant effect on neighbouring tissue.

The application of heat is inhibitory to the growth of cultures if the temperature be raised above the level of 44° C., but there is a remarkable resistance to the effects of cold. Tissues and strains of cells have been kept alive at a temperature of – 10° C. There is also a like power to resist exsiccation; one worker, by drying the heart of the axoloti and thus reducing it to 70% of its original bulk, kept it at a temperature of 4° C. for one hundred hours and then succeeded in making cultures from it. Pieces taken from the human heart or that of the chick have survived dehydration to about 80%; and the embryonic brain tissue of the chick, after exsiccation to 90·7% and a subsequent sojourn of 144 hours inside a refrigerator, has shown growth of nervous tissue after explantation.

Concerning the effects of irradiation, it is known that in chick fibrocytes or nerve fibres inhibition of growth follows exposure to ultra-violet, Hertzian and Gurvich rays. The influence of radium is not a matter of universal agreement, though Love has stated that cell sensitivity depends on the distance of the cell from its maturity. If the time since division is less than three hours, the sensitivity is constant; after three hours there is a noticeable decrease. Histologically, no determinations have as yet been made as to the irritative and lethal doses. The results obtained with X rays seem to correspond to those obtained with radium, and the premitotic phase seems to be that of greatest sensitivity.

From the chemical standpoint, growth is governed by the hydrogen ion concentration of the culture media, different tissues showing optimum growth with different concentrations. Nerve cells, for instance, migrate and grow at a pH of from 5·8 to 7·8, but their optimum concentration is between 6·6 and 6·8. Moreover, the explanted tissue itself has power to regulate the pH to this level. These facts probably explain the discrepancy in results obtained by various workers.

Perhaps the most promising work in the field of tissue culture is that upon the growth of viruses. As an example, chick embryo tissue in flask cultures has been inoculated with from 25 to 250 units of smallpox virus. In eight days the units increased four hundred fold, a proof that an explanted chick embryo can produce as much virus as a calf. This lymph has been used with success for the vaccination of infants. The viruses of herpes, of coryza, of Manchurian typhus and of yellow fever have now all been cultivated, and various types of Rickettsia virus have all been subjected to systematic analysis. The same is true of the viruses of psittacosis, foot and mouth disease, hog cholera and pseudorabies. Viruses productive of disease in rabbits have also been grown, as well as that causing grasserie, a pest in silkworms.

The literature concerning tumour culture is now very extensive and of absorbing interest. Sarcomatous tissue is much easier of cultivation than material from epithelial tumours, and the sarcomata of animals, such as chicken sarcoma and Crocker's and Jensen's rat sarcoma, may be explanted and transplanted without difficulty. The migration and growth are of two types: (i) that of amœboid macrophages and (ii) that of pure strains of fibroblasts.

The malignant fibroblasts of rat sarcoma resemble normal rat fibroblasts, except that their colonies are larger and their cells coarser and looser in arrangement. They show no other abnormalities and are not subject to degeneration, though slight physiological differences have been noted, especially in their propensity to liquefy a coagulum of rat plasma.

Two workers, Russell and Bland, have successfully explanted twenty gliomata, including medulloblastomata and astrocytomata. The migrating tumour cells showed great plasticity of form, but retained their morphological characters intact, proving a preservation of cell specificity and the justice of classifying tumours according to their cellular content. Since cancer cells are destructive of normal tissue, one experimenter explanted normal tissue simultaneously with material from a tumour. He reported having prevented liquefaction of the plasma and having successfully cultivated cancer cells for an unlimited period. While other workers have failed to corroborate these findings with cultures of carcinomata of the human breast and gall-bladder, there seems little doubt that animal cancers are more easily explantable in association with normal tissue. Various types of cancer differ in respect of fibrinolysis; mouse and rat plasmas liquefy more readily than chicken plasma, and there are small differences in the behaviour of spontaneous mammary tumours and Ehrlich's mouse carcinoma. Cancerous cells proliferate in a membranous manner. The number of mitotic figures does not vary from the normal, but chromosomes are less numerous. Sarcomatous and cancerous cells and tissues behave similarly in regard to glycolysis. Tumour cells do not grow better than normal cells under anaerobic conditions, but are indeed more sensitive and less resistant. Between the two types of cells there are only quantitative differences; no dogmatic statement as to biological differences is at present justifiable.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on June 3, 1936, DR. WALTER SUMMONS, the President, in the chair.

Physical Signs in Diseases of the Chest.

DR. C. H. FITTS read a paper entitled "Observations on Physical Signs in Diseases of the Chest" (see page 321).

DR. S. V. SEWELL congratulated Dr. Fitts on what was a delightful historical review of the subject; Dr. Fitts, though still a young man, had a very wide and concentrated experience of chest work and spoke with authority.

Dr. Sewell had been brought up in medicine in pre-skiagram days and had taught physical signs to many generations of students. It was only in the last twelve to fourteen years that he had found skiagrams of real value in the early diagnosis of pulmonary conditions. Even now he saw many skiagrams, mainly taken in the country, which were so bad as to be not only useless, but entirely misleading. These skiagrams usually showed the bony structure very clearly, and therefore showed no detail whatever of the lung structure.

Dr. Sewell would like to use as a text for what he wished to say, the skiagram shown by Dr. Fitts, in which a fairly large, definitely circumscribed area of consolidation had developed towards the apex of the left lung, which had shown no evidence of involvement in a skiagram taken a fortnight later. Such rapidly developing areas were quite common in adult tuberculosis, because they developed as the result of a spread and reinfection in an individual previously sensitized by a tuberculous infection, and corresponded to the results obtained years ago by Koch in his experiments on sensitized animals. These areas were largely due to an allergic reaction round a new area of infection and corresponded clinically to the attacks of so-called prolonged influenza, pleurisy and such, which were so universally related by patients coming for advice about well established tuberculous pulmonary lesions. These allergic areas produced definite physical signs and were of great significance in the early history of many pulmonary conditions. Dr. Sewell had had the opportunity of watching a number of patients with such patches. In favourable cases the whole allergic reaction cleared up in a month or six weeks, leaving a small patch of histogenous reaction or true tuberculous inflammation, which was most difficult to find on clinical examination and showed up as only a small persisting nodule in the skiagram. If the medical attendant was not alive to the frequency of these cases, they would continue to be undiagnosed and, as at present, the patients would be sent away for a short holiday, returning with improved general health, the allergic factor having cleared up, but the smouldering tuberculous focus still remaining.

Skiagrams were of great value in these cases, but it was most unwise to attempt to give a prognosis on one film taken from a patient who had recently recovered from a febrile disturbance. It was much better to wait until a second film was taken six weeks later. Dr. Sewell had been brought up in this work by that revered teacher, the late Sir Richard Stawell, and felt that a most careful physical examination of the chest was necessary in every case in which the patient complained of lassitude, loss of appetite and vague acid dyspepsia, with or without a little cough in the morning, often without expectoration. The investigation of such a case required not only an examination of the functional activity of the various parts of the lung before and just after coughing, but also repeated sputum examinations if sputum was obtainable. He had to admit that after the most careful clinical examination efficient skiagrams very frequently showed much more in the way of pulmonary involvement than the physical signs had led him to expect. Dr. Sewell had been visiting a sanatorium regularly for over twenty-five years and was forced to the conclusion that patients sent to the sanatorium showed that, in spite of improving radiological technique, cases were not being detected earlier than they were twenty years ago. He felt that this was largely due to a decline in the thoroughness in the search for early clinical signs. The cult of physical examination had reached its highest development twenty years ago. If clinicians were to advance in the art of physical examination and the interpretation of physical signs, then it behoved them to be thoroughly honest with themselves. They should examine carefully, commit their findings to writing and then correct and amplify them with the best possible skiagrams; in this way they would be able to advance the art of physical examination and to avoid looking upon radiological investigation as a short cut to diagnosis.

DR. W. J. NEWING congratulated Dr. Fitts on his very erudite paper; he had hoped that Dr. Fitts would have described in more detail the diagnosis of the conditions; but the critical survey that Dr. Fitts had made was long overdue. At the same time he felt that the ideal aimed at by Dr. Fitts, which would require that all doctors should have efficient radiographic apparatus and know how to use it, would be obtained only slowly, and at least until that time arrived accurate physical chest examination was necessary. In a recent address at the time of his retirement from hospital practice, Lord Horder had said that it was a mistake for the clinician to teach medicine: he should teach his students physical signs, and they could read the medicine for themselves. When a student had been found hard at work tapping out the borders of the heart to determine its size, he had been informed that his action was to be commended if he was doing it as a compliment to those who had been before him; but he was to be condemned if he was doing it to find out the size of the heart; he should go and examine the patient radioscopically. Dr. Newing pointed out that in reality the arts of auscultation and percussion were acquired only after years of practice; and if medical

students were given too ready access to the use of the screen he felt that this would be a retrograde step. Dr. Newing considered that in the case of a small child, if he could demonstrate the rise in pitch at a certain point, he could prognosticate the arrival of pneumonia without any necessity for radiography. The subject of bronchial breathing could be a perfect nightmare to a medical student, who would learn in his books that pneumonia, asthma and emphysema were all characterized by bronchial breathing. Dr. Newing thought that an attempt should be made to differentiate these physical signs. The tubular breathing of pneumonia indicating consolidation should be separated as an entity distinct from the bronchial breathing of asthma or emphysema. Dr. Newing pointed out that these vagaries of tone and pitch should be appreciated by students and should be explained to them; the student could get his own mental register only by long experience and concentrated use of the stethoscope. Dr. Newing congratulated Dr. Fitts for pointing to an ideal and stimulating those present to an effort to sift the bran from the chaff.

DR. S. O. COWEN said that, like the two previous speakers, he felt exercised in his mind by what Dr. Fitts had said so admirably; he felt disturbed as to the effect of these new doctrines on his own practice and from the point of view of clinical teaching. Two attitudes towards ordinary bedside clinical signs had been brought to their notice some two years earlier in that very room. Dr. Thomas and Dr. Hallam had presented a paper in which they had stated somewhat dramatically that these gods of student days had been knocked from their pedestals and that the bedside clinical signs were at their best time-consuming, clumsy, and in the main inaccurate contrasted with radiographic investigation. Dr. Cowen felt strongly that that attitude could not be reasonably advocated by a clinical teacher; he would rather take sides with Dr. Fitts and try to determine where he stood in the matter. The question of evaluation of the signs could be appraised from two standpoints: in addition to the practical clinical standpoint they could be regarded as scientific abstractions. Dr. Cowen doubted whether the physical basis of some physical signs was satisfactory, as it was taught at present. He recounted the experiences of Bendowe, who, in a sanatorium, had tried to detect cavities in three hundred and fifty patients; in less than 30% he was able to hear the cavernous breathing which was regarded as typical, though in another 50% of cases obvious auscultatory signs were present, but not those usually associated with cavitation. Another point raised by Dr. Cowen was the feeble nature of the usually accepted explanation of the frequent absence of breath sounds in early pneumonia. In defence of physical signs, Dr. Cowen referred to the misleading nature of skiagraphic reports in many instances of the development of empyema in the course of an ordinary attack of pneumonia, and stated that he would sooner depend on his own senses and the exploring needle. He admitted readily that there was an urgent need for revision of a good many of the current ideas on physical examination and its interpretation, but he thought that the final court of appeal should be the dead-house and not the shadows of the screen. They were not using to the best advantage the knowledge that they had; he agreed that the seeking for a sign—the short cut—was at the bottom of many of their failures.

Before concluding he would like to join issue with Dr. Fitts on an historical matter. Laennec thought that he had discovered in auscultation a method that would be exact, just as clearly as the finger or the sound gave information to the surgeon; that attitude was not right. Austin Flint, by 1876, had grasped the significance of the fact that very few signs were directly diagnostic of different diseases, and had stated the importance of keeping the physical signs in their proper places. Dr. Cowen deplored the fact that it seemed to be nobody's business to teach and to learn the proper methods of scientific approach; in the crowded medical curriculum no room had been found for training in scientific thinking; Dr. Cowen recommended the first part at least of Karl Pearson's "Grammar of Science" to anyone who wished to know more about the methods of gathering evidence on scientific lines. Dr. Cowen felt that clinicians were not on very sure ground in their interpretation of physical signs or of precisely the proper place that physical signs should occupy in diagnosis.

DR. J. G. HAYDEN, after expressing his appreciation of Dr. Fitts's address, said that a most important question had been raised, which involved a decision as to whether they were to abandon what they had been taught; physical signs were of very doubtful assistance in tuberculosis, but were very helpful in most other diseases of the chest. It was the site of tuberculous infection which placed them in difficulty. Some of the eponymic signs were ridiculous and useless, and should be jettisoned; they learned that amphoric breathing was the sign of pneumothorax, but experience with artificial pneumothorax had taught them that absence or diminution of breath sounds was the usual finding; again, breath sounds were by no means always absent in empyema. Dr. Hayden stressed the importance of taking every factor into consideration, and said that the evaluation of the signs was the important thing. He referred to a valuable small pamphlet containing three lectures by Dr. Robert Hutchison on diagnosis, prognosis and treatment. Dr. Hayden considered that the skiagram was the best single factor in the diagnosis of early tuberculosis; he recalled that six years earlier Sir Richard Stawell had said that the older he got, the more faith he was placing in skiagrams, and had advised Dr. Hayden to send patients to the radiographer when the history was suggested but the expected physical signs were not present.

At the same time Dr. Hayden considered that skiagraphy was doing a lot of harm; faulty interpretation at times led to the diagnosis of early tuberculosis when it was not present; a skiagram was useless soon after an hæmoptysis, when there really might be only a very small apical lesion; it should not be forgotten that lipiodol stayed in the chest for two years or more and in subsequent radiograms might cast a shadow very suggestive of those of tuberculous infiltration. Persistent evening rise in temperature indicated only the presence of some infection, which was by no means necessarily tuberculous. In three instances in his experience patients had been classified as tuberculous who had been proved subsequently to be suffering from *Brucella abortus* infection, cholecystitis and appendicitis respectively. He would also like to issue a word of warning against the pronouncement of the diagnosis of tuberculosis when it was based chiefly on the identification in a stained specimen of sputum of one or two organisms said to be the bacilli of tuberculosis; it had even been proved that the acid-fast bacilli might belong to the water supply. He had been very impressed with the demonstration Dr. Fitts had given of the use of lipiodol for outlining the extent of an empyema cavity, and thought that this was a very valuable method. He would also like to mention its use in bronchial fistula cases; by this means it had been shown that when basal tuberculosis spread to the other side the lipiodol ran into the lower and middle portions of the opposite lung; it was important, therefore, that such patients should sleep on the side affected.

DR. J. BELL FERGUSON said that it was some twenty-seven years since he had first learned his physical signs. He thought that Dr. Fitts had performed a very useful duty; it would be necessary for those present to decide what they were going to do about the teaching of medical students. Dr. Ferguson suggested that in many cases of chest trouble a medical man had to discover the nature of the disease without the aid of skiagraphy; so that the student would have to be put through his physical signs just as carefully as formerly. Nevertheless he must learn to suspect tuberculosis in the early stages and should obtain a skiagram, if possible, instead of waiting for the development of unequivocal physical signs. It was a very valuable exercise to let students hear a description of the physical findings elicited by an expert, followed by a demonstration by a radiologist which would draw attention to the inaccuracies; such a combined demonstration was carried out in his department each Saturday morning. Dr. Sewell had pointed out the importance of close attention to the history-taking in early tuberculosis, and Dr.

Bell Ferguson confirmed this view. When examining nurses and other young adults physically it was of first importance to obtain radiological evidence before passing them as free from chest trouble. Dr. Sewell had also pointed out the allergic response and the rapid spread which occurred. Dr. Ferguson was surprised that no one had suggested the use of injections of tuberculin, especially when radiological investigation was not available or was not helpful; he would strongly urge that tuberculin testing should be used.

DR. K. H. HALLAM said that he too, like Dr. Fitts, had a creed built up of personal experience obtained in ten years of general practice, followed by intensive study in the practice of radiology; in general practice on many occasions he had missed the early diagnosis of tuberculosis because of the inadequacy of the information supplied by the physical signs elicited. In the diagnosis of chest conditions he would rely on the information obtained in taking the history and on the results of radiological investigation, and would place the physical examination a bad last by comparison. He questioned, therefore, the place given to physical signs in the teaching of medicine. Dr. Hallam recalled that at school the subject of history could be learned easily, whereas the binomial theorem was learned only laboriously; it was true that to acquire knowledge one had to work hard, whether the knowledge acquired was useful or otherwise; he would like to draw an analogy between the subjects in the school curriculum on the one hand and the ease of history-taking and the laborious search for physical signs on the other. He would classify the binomial theorem and the physical signs together as examples of mental gymnastics. He thought that students should be told that the physical signs must be discounted to get the truth. Dr. Hallam wished to make it quite clear that in making these strictures he was of the opinion that it was not the man that was at fault, but it was the method. It should be recognized too that there were extremes of evaluation in radiology; radiology was of little value in the examination of contacts of tuberculous patients and also when a decision was to be made concerning the superimposition of tuberculosis on pneumonokoniosis. As a guide to which patients should be examined radiologically, physical signs, taken with the history, were of great importance.

DR. J. F. MACKEDDIE said that he belonged to the old brigade, but he congratulated Dr. Fitts on a very erudite and practical paper, and would not quarrel about the value of radiology in the early diagnosis of tuberculosis. He recalled that fourteen years ago, when he was at Brompton, the doctors would not presume to certify the presence of an early tuberculous lesion on skiagraphic evidence unsupported by physical signs. With modern improvements in radiology he did not doubt that nowadays they would probably say "yes" where they would then have said "no" on physical signs. Dr. Mackeddie commented on the inconsistency of the nomenclature of physical signs and pointed his remarks by reference to the weird and wonderful things that students said they heard, and he gave an amusing account of the rabbit and bed-pan incident. He then referred to the importance which old-time physicians placed on the general condition of the patient, and the acuity with which they observed divergences from the normal outward appearance, and he expressed the hope that such physical methods of examination would be perpetuated.

DR. W. OSTERMEYER emphasized the importance of clarity of thought in drawing inferences from the facts observed at a physical examination; the physical signs and their interpretation were physical problems only and were dependent on the properties of the underlying structures; it was legitimate to deduce from them only the physical condition of these structures, and one should not evaluate the physical signs medically or diagnostically.

DR. WALTER SUMMONS thanked Dr. Fitts for his scholarly and provocative address, and reminded those present that all were still students and that the practice of medicine was at no time fixed, but was constantly fluctuating. Sir Richard Stawell, in his heyday as a teacher, had taught them how to interpret physical signs, but they had to be prepared to add new methods of proven value to their armamentarium as they became available. He also commented on the constant changes in shades of meaning of English words; many of the speakers that evening had used the word "evaluation" in the sense that used to be filled by the word "interpretation".

Dr. Fitts, in reply to the discussion, said that he could answer a lot of questions by describing a Tuesday evening round with his chief at Brompton. Dr. Fitts used to meet his chief at the door of the hospital at five o'clock and would be asked how many new patients had been admitted since the previous visit. Dr. Fitts would commence to enumerate them, but would be stopped from referring to diagnosis. At the bedside he would be closely cross-examined concerning the history of the patient, and then the chief would proceed to make a careful physical examination, with the patient sitting up at first and again with the patient lying down. Complete notes of the findings would be recorded and afterwards compared with the notes that Dr. Fitts had made when he examined the patient. A discussion would follow to explain any discrepancies that might exist in the two reports, and finality would be reached respecting the physical signs. They would then call to them the nurse bearing the viewing-box, who had been trailing in the rear, and would compare the physical findings with the radiological appearances. Dr. Fitts went on to refer to the old tag that the science of today was the rubbish heap of tomorrow, to support his view that they should not be dogmatic. He deplored the tendency of some clinical teachers to take the physical signs out of their context, and also drew attention to the deleterious effect of the scarcity of chest cases for teaching purposes in the public hospitals in Melbourne.

Post-Graduate Work.

POST-GRADUATE COURSE IN TUBERCULOSIS.

THE New South Wales Post-Graduate Committee in Medicine announces that a post-graduate course in tuberculosis, its diagnosis, prognosis and treatment, will be held from October 26 to November 13, 1936, as follows.

Monday, October 26, 1936.

4 to 5 p.m.—Lecture on the public health aspects of tuberculosis, organizations under the Board of Control, the responsibilities of practitioners, notification, contacts *et cetera*, by Dr. H. G. Wallace, Director, Tuberculosis Division of New South Wales.

5 to 6 p.m.—Cinematograph demonstration, by courtesy of the Director-General of Public Health.

Wednesday, October 28, 1936.

4 to 5 p.m.—Lecture: "The Examination of Contacts and Familial Examinations in Tuberculosis by the General Practitioner", by Dr. John Hughes, Medical Officer, Tuberculosis Division.

5 to 6 p.m.—Lecture: "What the General Practitioner Should Know Concerning Silicosis", with pathological and radiological demonstrations, by Dr. Charles Badham, Director, Division of Industrial Hygiene.

Friday, October 30, 1936.

3.30 p.m.—Lecture: "The Early Diagnosis of Pulmonary Tuberculosis", by Dr. S. A. Smith.

4.30 p.m.—Lecture: "The Differential Diagnosis of Pulmonary Tuberculosis", by Dr. Allan Walker.

5.30 p.m.—Lecture: "The X Ray Diagnosis of Pulmonary Tuberculosis", by Dr. H. R. Sear.

Monday, November 2, 1936.

4 p.m.—Lecture-demonstration: "The Pathology and Bacteriology of Tuberculosis", by Dr. A. H. Tebbutt.
5 p.m.—Lecture: "Treatment and Prognosis in Pulmonary Tuberculosis", by Dr. Cotter Harvey.

Wednesday, November 4, 1936.

2.15 p.m.—Royal Prince Alfred Hospital, Anti-Tuberculosis Clinic: Demonstration of the work of the clinic, with special reference to skin tests, cases illustrating the selection of cases for sanatoria and prognosis, especially in relation to cavitation, by Dr. Allan Walker, Dr. W. A. Bye and Dr. A. W. Morrow.

Friday, November 6, 1936.

4 to 4.30 p.m.—Lecture-demonstration: "Pleurisy and its Treatment", by Dr. Wilfred Evans.
4.30 to 5 p.m.—Lecture: "Hæmoptysis", by Dr. E. H. Stokes.
5 to 5.30 p.m.—Lecture: "Surgery of Pulmonary Tuberculosis", by Dr. M. P. Susman.
5.30 to 6 p.m.—Lecture: "The Diagnosis of Genito-Urinary Tuberculosis", by Dr. R. Bridge.

Monday, November 9, 1936.

4 to 6 p.m.—Royal Alexandra Hospital for Children: "Medical and Surgical Tuberculosis in Children", with demonstration, by Dr. Wilfred Vickers and members of the staff.

Wednesday, November 11, 1936.

2.30 p.m.—Royal North Shore Hospital, Anti-Tuberculosis Clinic: Demonstration of cases illustrating difficulties in diagnosis and demonstration of Mantoux sedimentation test, by Dr. Cotter Harvey and Dr. Bruce White. Lecture-demonstration: "Tuberculosis of Ear, Nose and Throat", by Dr. E. P. Blashki.

Friday, November 13, 1936.

4 p.m.—Special questions and discussion—problems arising and consultations.

All lectures and demonstrations will be held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, except where otherwise indicated. The course will be held only if eight entries are received before Monday, October 12. Dr. W. L. Calov will act as supervisor of the course.

The fee for the course is £3 3s. Exchange should be added to country and interstate cheques.

Applications should be made as soon as possible to the Secretary, New South Wales Post-Graduate Committee in Medicine, The University of Sydney, Sydney.

Correspondence.

HISTIDINE TREATMENT OF PEPTIC ULCER.

SIR: Yesterday, among some magazines in a rack, an unopened copy of THE MEDICAL JOURNAL OF AUSTRALIA presented itself. Frenziedly tearing the wrapper, for I am punctilious in at least opening my medical journals, I discovered to my horror that the issue was dated June 20, 1936, and that it contained a courteous query from Dr. C. Stanton Hicks, of Adelaide University, as to a case reported by me. The diagnosis of duodenal ulcer had been made by a radiologist, and the symptoms had apparently been aggravated by histidine injections and greatly relieved by "Belladonna Neutralon".

The particulars which I am able to furnish are as follows:

Radiologist's report. Stomach: High in position, with transverse lie. Increased peristalsis and tone. Normal mobility and flexibility. Curvatures free of any defects. No residue after six hours, at which time the head of the meal had entered the descending colon—hypermotility.

Duodenum: The first part or cap shows a persistent deformity due to ulceration.

Small bowel: No abnormality was detected.

Summary: The findings are indicative of an ulcer of the duodenum, not associated with any obstruction of the pylorus, but accompanied by increased motility of the stomach.

Occult blood: It was not considered necessary to test for this, as there had been a slight but definite melena about six weeks before the screening.

Fractional meal: Not done. A titration of the stomach contents two hours after a test meal showed free hydrochloric acid. The pain was always temporarily relieved by alkalis, and carbonates caused copious eructations of gas.

This comprises all the information that I am able to give in direct reply to Dr. Hicks's queries, and to me the evidence of ulcer seems almost complete, but the following additional items may be of interest.

The patient's appendix was removed some years ago. There is no indication of dental or other sepsis. As regards the subjective nature of the symptom complained of—nocturnal pain—the patient received the histidine injections under the impression that here at last was something more than a mere palliative, something really curative. He was in fact eager for the treatment. After a few injections he was, to his great surprise, awakened by epigastric pain, which appeared to get worse as the injections continued. In spite of this, he persevered up to the twelfth dose.

The "Belladonna Neutralon"—a sample tin—was administered rather after the manner of "well, try this and see if it does you any good", and there was immediate relief, which has continued for months. This would indicate that the pain was in no way influenced by the patient's mental attitude.

Dr. Stanton Hicks states that my "conclusions are of more than ordinary significance for us all". Permit me to point out that my letter did not purport to expound any general conclusions about the matter, but merely to record the facts, as known to me, about this particular case.

Yours, etc.,

H. G. LOUGHRAN.

Kyneton, Victoria,
August 20, 1936.

A SECTION OF CHEST DISEASES.

SIR: I would be grateful if you would make available the contents of this communication.

At a meeting of the Radiological Section of the British Medical Association (Victorian Branch) on Tuesday, July 21, it was suggested that a Section of Chest Diseases be formed.

It is probable that many who did not attend that meeting would be interested in this project, and I would ask all members of the Branch who favour this proposal to forward their names to me at their earliest convenience; should sufficient support be indicated, the necessary steps for the formation of the section will be taken.

Yours, etc.,

DAVID B. ROSENTHAL,
Medical Superintendent.
Gresswell Sanatorium.

July 22, 1936.

Books Received.

MINOR SURGERY AND THE TREATMENT OF FRACTURES (HEATH, POLLARD, DAVIES), FOR THE USE OF HOUSE SURGEONS, DRESSERS, AND JUNIOR PRACTITIONERS; Twenty-First Edition; by G. Williams, M.S., F.R.C.S.; 1936. London: J. and A. Churchill. Crown 8vo, pp. 493, with illustrations. Price: 10s. 6d. net.

MEDICAL ASPECTS OF CRIME, by W. N. East, M.D., F.R.C.P., with foreword by the Right Hon. Sir John Simon; 1936. London: J. and A. Churchill Limited. Demy 8vo, pp. 447, with illustrations. Price: 18s. net.

A NEW DICTIONARY FOR NURSES, by L. Oakes, S.R.N., D.N.; Fourth Edition; 1936. Edinburgh: E. and S. Livingstone. Demy 16mo, pp. 388, with illustrations. Price: 3s. net.

CHANGE OF LIFE IN MEN AND WOMEN, by M. C. Stopes; 1936. London: Putnam and Company Limited. Crown 8vo, pp. 296. Price: 6s. net.

Diary for the Month.

SEPT. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 9.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 11.—Queensland Branch, B.M.A.: Council.
SEPT. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 15.—Tasmanian Branch, B.M.A.: Council.
SEPT. 16.—Western Australian Branch, B.M.A.: Branch.
SEPT. 17.—New South Wales Branch, B.M.A.: Clinical meeting.
SEPT. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 23.—Victorian Branch, B.M.A.: Council.
SEPT. 24.—South Australian Branch, B.M.A.: Branch.
SEPT. 24.—New South Wales Branch, B.M.A.: Branch.
SEPT. 25.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. E. H. Barrett has been appointed Certifying Medical Practitioner at Swan Hill, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. J. M. Bonnin and Dr. S. L. Seymour have been appointed Resident Medical Officers at the Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. R. S. Rogers has been reappointed President of the Medical Board of South Australia, under the provisions of the *Medical Practitioners Act*, 1919.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages XX-XXII.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer.
BLAIR ATHOL DISTRICT HOSPITAL, QUEENSLAND: Medical Officer.
BROKEN HILL AND DISTRICT HOSPITAL, NEW SOUTH WALES: Resident Medical Officer.
KALGOORLIE DISTRICT HOSPITAL, KALGOORLIE, WESTERN AUSTRALIA: Resident Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.
NEW SOUTH WALES MASONIC HOSPITAL, ASHFIELD: Resident Medical Officer.
ROYAL AUSTRALIAN NAVY: Medical Officer.
SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Instructor in Practical Anæsthetics.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Honorary Officers.
THE OTAGO HOSPITAL BOARD, DUNEDIN, NEW ZEALAND: Resident Surgical Officer.
THE UNIVERSITY OF ADELAIDE, SOUTH AUSTRALIA: Elder Professorship of Anatomy and Histology.
THE UNIVERSITY OF MELBOURNE, VICTORIA: Stewart Lectureship in Medicine.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officers, Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
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